The Journal of the American Association of Nurse Anesthetists

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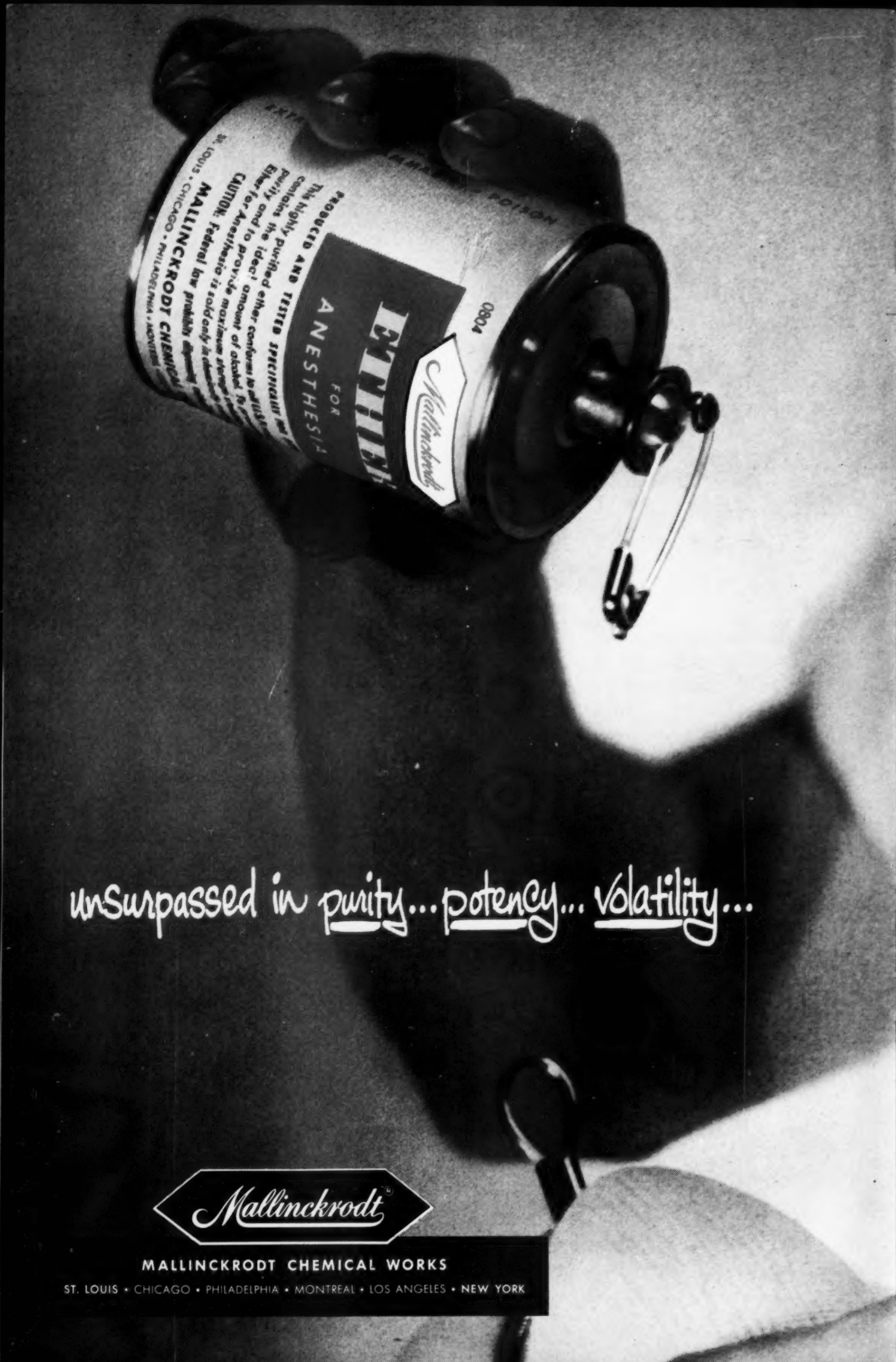

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The Journal OF THE American Association of Nurse Anesthetists
Vol. XXI No. 3 August, 1953

New Experiences - Old Friendships

The American Hospital Association again welcomes the American Association of Nurse Anesthetists at its convention in San Francisco. This is our 55th Annual Convention and the twenty-first at which we have had a joint meeting with your association.

San Francisco—one of the most beautiful cities in the world—is a favorable spot for our meetings. It is very difficult not to speak of all the recreational opportunities there and, for those of us who live in the East, of the many parks and scenic wonders between home and the West Coast.

Conventions are an American tradition and the large attendance they attract is quite contrary to anything found elsewhere in the world. It is a time to combine business and pleasure; to make new friends and to renew old friendships. Yet, the serious scientific programs of our two associations clearly justify the time and money invested attending the convention. Through no other method is it possible to review annually all the new developments in the hospital field.

The American Hospital Association has been very close to your association throughout the years. Your headquarters was moved into the American Hospital Association building in 1937 and only as both organizations grew did lack of space force a separation in 1951. This separation in no way indicates any less interest on the part of this association or its member hospitals in the programs to improve the quality of service rendered by your members within the hospital field.

The hospital merchandise mart at the San Francisco convention again will be the largest exhibit of hospital supplies and equipment shown in any one place. This year the American Hospital Association will have a large pavilion as an addition to the San Francisco Civic Center to accommodate those who wish to exhibit. Nurse anesthetists will find the exhibit of anesthesia equipment and supplies very complete and informative and, as usual, an important part of the educational experience of the annual convention.

San Francisco will give opportunity for our members on the West Coast to participate more actively in the annual convention, all in the interest of better preparation for those who work in the hospital field and who carry such serious responsibilities for the provision of good care to the patients we serve.—George Bugbee, Executive Director, American Hospital Association.

Electrostatic Explosion Controls in Hospital Operating Rooms

Robin Beach, Fellow A.I.E.E.*
New York

Static electricity as generated in hospital operating rooms is, indeed, of no different parentage than that which haunts industry and, by its sabotage, causes costly and devastating fires and explosions. However, in hospital operating rooms, more causes for electrification occur within comparatively small areas than in almost any industrial plants. Also more hazards of potential explosions exist here than in many types of industry.

Because of the administration of pure oxygen mixtures rather than air with anesthetic products through rebreathing systems to anesthetized patients, explosive ranges are greatly broadened and explosions, when they occur, are intensified in the relatively small operating rooms. When explosions take place, generally at least one life is sacrificed—that of the patient—and, sometimes, several injuries and destruction of physical plant result.

How, why, and where these electrostatic properties display themselves in hospital operating rooms and the philosophy of techniques for controlling the hazards are the objectives of this paper.

Read before the Middle Atlantic Assembly of Nurse Anesthetists May 20, 1953.

Revised text of a paper, "Electrostatic Safety for Hospital Operating Rooms," published in Electrical Engineering, Volume 72, April 1953, and reprinted by permission of American Institute of Electrical Engineers.

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How Electrostatic Sparks are Created

Static electricity is brought into being by the contact of one substance with another. This process by which electrification of the two substances is created at their contacting interface is know as "contact electrification." When in contact, unbalanced interatomic forces across the interface cause electrons to migrate from the substance of higher dielectric constant through the myriad of intervening contacting points to the substance of lower dielectric constant. This phenomenon occurs between all pairs of contacting substances whether they possess conductive or dielectric properties.[1]

The substance receiving the electrons thus acquires an electrification of negative polarity while the substance from whence the electrons migrate possesses an equal electrification but of positive polarity.

The differences of potential across interfacial boundaries which are attained for pairs of contacting substances, in rare instances only, reach a maximum of one volt; mostly, the voltage thus developed acquires an order of millivolts and, commonly, even of microvolts.

1. Beach, R.: Static electricity in industry, Electrical Engineering **64**:184-194, May 1945.

When two contacting substances which are being separated are conductors, all free electrons on the one surface at the interface return from whence they came before the last point of contact is broken. Hence, the conductors retain no electrification.

For insulators, the electrons which are acquired by the one surface in contact with the other are so effectively impounded by their lack of migratory freedom that they are quite effectively entrapped during the process of separation. Hence, if one or both of the substances are excellent insulators, following their separation they each are electrified and each displays the characteristics of respective positive or negative electrification.

No substances are perfect insulators, hence the speed with which the contacting substances become separated comprises a determining factor in how many electrons are entrapped; and, quite generally, the electrification attained from rapid separation of the two substances is materially increased. Within certain limits the electrification of a substance during separation from another substance increases in linear proportion with the increasing speed of their separation.

The electrified surfaces of two substances in so-called contact comprise a capacitor and these surfaces, on either side of the interface, always possess opposite polarities.

As the two electrified surfaces at the interface are separated and assuming the electrification to remain at constant magnitude, the voltage between the surfaces increases directly with the distance of separation. In this manner, potential differences of thousands, tens of thousands, or even hundreds of thousands of volts may be developed between the separating surfaces.

Were it not for the two mitigating factors of charge conduction leakage and ionization loss of charge in the behavior of the static electricity as the interfacial surfaces separate in this manner, the voltage across the lengthening gap between the separating surfaces would increase indefinitely. This, of course, it does not do.

In industrial operations, both of these discharging processes, together, react to restrain and circumscribe the voltage rise to some degree as the separation of the electrified surfaces increases.

Fire and explosion hazards are the most serious electrostatic problems, and these hazards exist when electric sparks are created in working areas where vapors of flammable products are present. Electrostatic sparks are most commonly initiated by induction where one of the electrified substances approaches a grounded conductor to which it sparks.

By induction, electricity of opposite polarity to that of the nearby electrified substance appears on the grounded conductor. If the voltage between the two exceeds the breakdown potential gradient of the air, a spark occurs. The ignition energy in the spark is a function of both the magnitude of the induced voltage across and the capacitance of the two oppositely electrified electrode areas.

The energies in electrostatic sparks necessary to ignite many flammable mixtures are so small that they are designated in tenths to even thousandths of a millijoule, a millijoule being a thousandth part of a joule or watt-second of energy.

Electrostatic sparks of ignition intensity for flammable vapor-air or vapor-oxygen mixtures may be created by the flip of a sheet on an operating table, by the handling of a rubber tube or re-breathing bag, by sudden spurting of gases through orifices from their cylinders by improper manipulations of valves, by the rubbing of gar-

ments on a stool in certain instances, or by walking on improperly constructed or conditioned floors. Igniting sparks in hospital operating rooms may be caused by differences of potential as low as 1100 volts.

Critical Elements in Hospital Operating Rooms

In principle, the elimination of electrostatic hazards in operating rooms resolves itself to the provision of suitable conductivity into each and every facility within the operating room, including especially the flooring and the clothing and footgear of the personnel. In regard to flooring, moderately conductive properties are preferred to high conductivity. With conductive qualities provided throughout, no accumulation of static electricity can exist on facilities within hospital operating rooms; and, hence, no electric sparks can occur to ignite ambient flammable mixtures.

In practice, such provisions of conductivity within operating rooms, while recognized as essential in implementing the inexorable tenets of safety and thus in eliminating all electrostatic hazards, seem not to be regarded as gravely as the jeopardy of the situation requires.

The safety provision that operating room personnel unfailingly change into cotton garments throughout and use conductive footgear before entering the hazardous areas of operating rooms should constitute an inviolate regulation of every hospital and one that should be demanded of each and every member of the operating staff.

Although conductive rubber coverings on mattresses and conductive rubber sheets and pads for operating tables are available and extensively used, these and other associated facilities, such as sheets, pillow cases, and blankets, when dry, may generate dangerous potentials when they are flipped on or off operating tables in the many ways practiced by nurses and doctors.

Sheeting and other cotton goods used on operating tables should be rendered reasonably conductive by some acceptable means. Unless these problems are resolved, operating tables will continue to remain a focal center of incipient hazards within hospital operating rooms.

The rubber tubing, re-breathing bags, and other such specialized appurtenances used in the re-breathing techniques of applying anesthesia are still largely dielectric and, as such, they constitute particularly susceptible instrumentalities for the generation of dangerous electrostatic potentials. Rubber facilities seem not to be sufficiently developed as yet, conductive-wise, to meet satisfactorily the exacting requirements of anesthetists in providing superior anesthetizing service and enduring conductivity. Also the rubber gloves used by surgeons in operating rooms, until made conductive, will doubtless continue as incipient sources of electrification.

The practice by surgeons and others of dusting into their rubber gloves talc powder and other dispersions which are highly dielectric should not be permitted in operating rooms. Films of talc, accumulating on the floor, develop sufficiently high resistance to render semi-conductive flooring almost non-conductive and, therefore, hazardous.

Most of the facilities in operating rooms, other than the operating table, are portable and are provided with casters or rubber leg tips. The casters and leg tips, of course, are composed quite generally of conductive rubber with the thought that their contact resistance to floor is low. However, this is commonly far afield from factual evidence and, in particular, talc has been found to increase contact resistances to dangerously high values.

Drag chains of various types, attached to portable equipment for the alleged purpose of providing grounding contact with flooring, have been shown by repeated tests to possess little, if any, value as a grounding agency. The separation of the dangling links constitute an ignition-spark hazard during electrostatic discharge; and, in the interest of safety, the use of such chains should be abandoned.

A positive and effective grounding device, now being developed, will soon be on the market to replace drag chains in hospitals, as well as to eliminate the contact resistance hazards of casters and leg tips regardless of their composition.

Safety Floors in Operating Rooms

The scientific philosophy which is basic to the recommended use of conductive safety floors in hospital operating rooms stresses the provision of conductive intercoupling between all operating-room equipment and personnel for the elimination of any differences of electrostatic potential.

Proper intercoupling by appropriate and well-maintained conductive flooring is the priceless key to effective control of almost all of the electrostatic hazards currently existing in hospital operating rooms. The NFPA Committee on Hospital Operating Rooms is to be complimented on its propitious recognition and adoption of conductive flooring for the resistance-controlled ground plane to serve as the intercoupling agency between all floor-borne equipment and personnel.

In order to translate into safe practice this basic concept of NFPA, however, certain factors relating to it require close analytical scrutiny and sound engineering judgment, born of specialized experience and related research. Some of these pertinent safety factors are:

1—upper and lower safe resistance limits for operating room floors;
2—problems in measuring the resistance of operating room floors;
3—important characteristics of homogeneous resistance for floors; and
4—use of alarm signals as an educational requisite in detecting and controlling electrostatic hazards.

Each of these safety factors is now considered under its related caption.

Protective Resistance Limits for Floors

The upper and lower safe resistance limits for hospital operating room floors are somewhat critical; and, the most effective control of the fire and explosion hazards in hazardous areas is contingent not only on the floors possessing resistance within relatively narrow upper and lower safe limits but, also, on maintaining the cleanliness of the floors for the purpose of preserving the desired range of resistance, once it is attained.

The lower the floor resistance is made the more effectively electrostatic potentials and their spark hazards would appear to be controlled—actually to their elimination. However, low resistance flooring, while appearing to possess most desirable properties in this respect, fortunately is not necessary, electrostatic-wise; and, unfortunately, low resistance flooring introduces its own peculiar brand of otherwise serious hazards.

If the live or "hot" wire of an electric cord or electrical appliance becomes exposed accidentally and makes contact with conductive flooring and, thus, with its intercoupled equipment and personnel, the surgeons and attendants in being electrified could receive electrical shocks, possibly with grave circumstances to themselves or the patient, by touching any grounded object.

Although the threshold alternating current for lethal electric shock lies in the order of 100 milliamperes, mild electric shocks, which may cause flinching from sudden muscular responses, especially of the fingers, occur at current values as low as one milliampere. Prudent judgment relating to safety considerations, therefore, prescribes the limitation of threshold shock current, by resistance control, to not more than one milliampere. For 125-volt electric service, the threshold control resistance is thus indicated in the value of 125,000 ohms between any part of the floor and ground or through the floor between spaced electrodes.

In addition to electric shock hazards to operating room personnel, an exposed live wire may cause electric arcs of sufficient heat energy, under conditions of low resistance flooring, to ignite ambient flammable mixtures. The threshold ignition energy of this potential arc ignition is controlled by resistance, incorporated homogeneously into the flooring, through a limiting resistance in the arc circuit to ground in the order of 100,000 ohms.

Hence, to guard against electric shock hazards to operating-room personnel and to eliminate the ignition hazards of electric arcs, both of which hazards originate from exposed live wires of 125-volt electric service, the threshold safety resistance between floor and ground or through the floor between spaced electrodes is about 125,000 ohms.

Applying a reasonable factor of safety to the above value of threshold safety resistance of flooring or of flooring to ground, the lower safe limit of this resistance is indicated in the order of 250,000 ohms rather than the value of 25,000 ohms recommended in NFPA Bulletin No. 56.[2]

2. NFPA No. 56: Recommended safe practices for hospital operating rooms. National Fire Protection Assn., 60 Batterymarch St., Boston 10, Mass.

In Bulletin No. 56, NFPA calls attention to the use of insulating transformers, with their control and signal systems, as a means of eliminating potential shock or arc-ignition hazards throughout operating rooms and other hazardous hospital areas by providing a separately-installed ungrounded electrical system. However, the proposed floor and floor-to-ground resistance of 250,000 ohms, as a lower resistance limit, provides protection against these electric shock and arc-ignition hazards without any additional cost and without such special facilities with their initial costs and their continuing maintenance expenses.

The safe upper limit of floor resistance is established by the time required for the discharge through floor of the highest source of static electricity in the operating room. If this discharge time is less than the time required to accumulate the generated static electricity, the electric charge is then dissipated as rapidly as it is generated and no appreciable differences of potential can occur.

In this regard, the research studies of Mr. Paul Guest and his associates at the U. S. Bureau of Mines on the charging and discharging rates for large capacitors,[3] such as insulated hospital operating tables, lead to their conclusion "Provision for removing aggregate charges in 0.01 second, or 15 to 30 times faster than they normally can be developed in anesthetizing locations, would seem to give enough margin of safety." These studies also disclosed that floors with a resistance to ground of 50 megohms are safe from electrostatic spark hazards for the largest capacitors found in hospital operating rooms under the maximum charging rates known to occur there.

3. Guest, P. G.; Sikora, V. W., and Lewis, B.: Static electricity in hospital operating suites: direct and related hazards and pertinent remedies. Bureau of Mines Report of Investigations 4833, p. 42.

Reference to Figure 1 explains the significance of the time constant in controlling the charging or discharging rate of a capacitor through a resistor, as shown by the electrical circuit insert.

Thus applying a reasonable safety factor to a floor resistance of 50 megohms, which is already determined to be safe, the upper safe resistance limit for hospital operating room floors to ground or between 3-foot spaced electrodes is conservatively indicated in the order of 10 megohms rather than the one megohm value recommended in NFPA Bulletin No. 56.

No equipment measured to ground should exceed the proposed upper safe resistance limit of 10 megohms. The contact resistances of floor-borne equipment preferably should not exceed the proposed lower limit of floor resistance for maximum safety.

After the floors of operating rooms have been provided with ground and surface resistances within the upper and lower safe limits, as explained and recommended, they can be rendered electrostatically dangerous, nevertheless, by the subsequent and ill-advised use of insulating coatings of polishing wax or by inadvertent accumulations

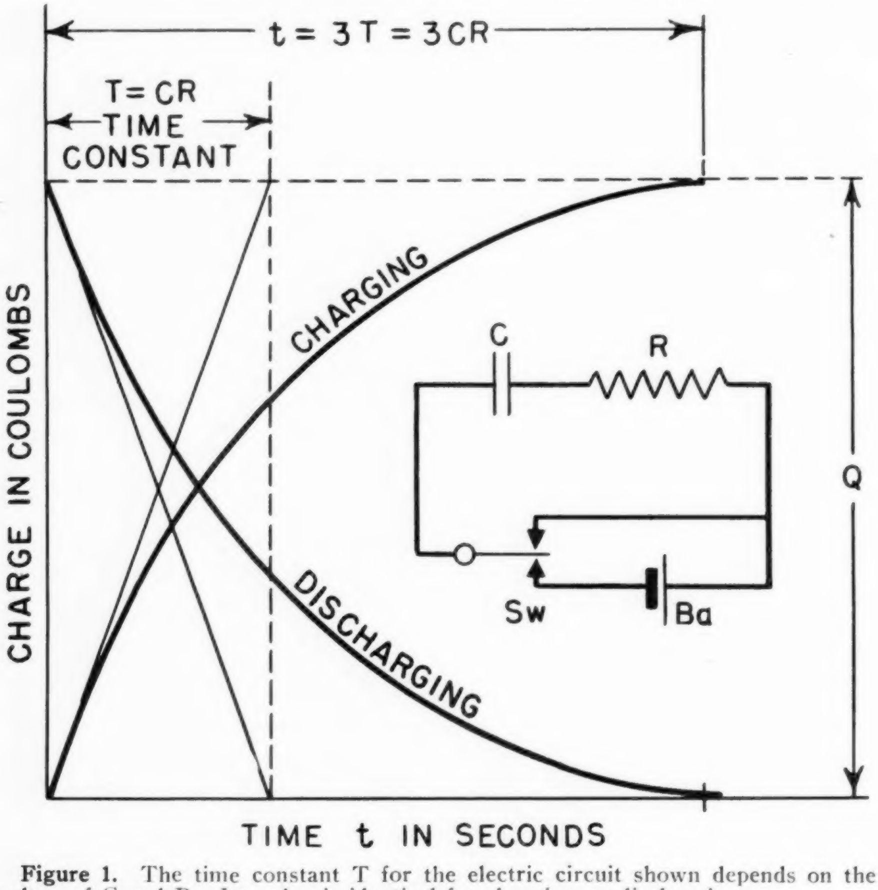

Figure 1. The time constant T for the electric circuit shown depends on the values of C and R. Its value is identical for charging or discharging.

of talc and other forms of insulating dispersions. However, floor dressings which maintain the floor resistance within the prescribed limits may be safely used, if desired.

In addition, the floors, after being provided with safe upper and lower limiting resistances, may be rendered dangerous from electric shock and arc-ignition hazards by the subsequent and ill-advised spilling of water or other conducting agents on the floor or by throwing wet towels and wet sheets onto the floor or by placing wet sheets around the bases of operating tables or anesthesia machines presumably for discharging static electricity—an unnecessary and inherently hazardous practice.

Problems in Measuring Floor Resistance

The five-pound electrodes, recommended by NFPA for measuring the resistance of floors, apply a pressure of one pound per square inch (1 psi) on the floor surface. NFPA also recommends that between the two 3-foot spaced electrodes, a resistance-measuring instrument should be employed which applies a 500-volt difference of potential between them. These specifications seem ill-advised both as to the light contact pressure of electrodes on the floor and as to the voltage applied for measuring floor resistance under conditions which exist in hospital operating rooms.

Almost any equipment supported on the floors of operating rooms, including even stools and light-weight tables, because of their small contact areas with the floor, yield high contact pressures which range from over 100 psi for stools to as high as 1000 psi for operating tables. Hence, test electrodes with a contact pressure of only 1 psi possess no commensurate relationship with the high contact pressures of the variety of floor-supported equipment, such as employed in operating rooms.

In addition, a 500-volt instrument for measuring floor resistance between the two 3-foot spaced electrodes, as recommended by NFPA, does not impress a test voltage which is commensurate with the magnitude of electrostatic voltages involved in threshold sparking hazards as they exist in operating rooms. For such resistance measurements, an instrument rated at 1000 volts is indicated.

This statement is predicated on the well-known practice in the technique of measurements of utilizing measuring parameters which simulate as closely as possible those which exist in the field. Hence, since an electrostatic potential of about 1100 volts has been determined as the threshold value for creating ignition sparks in hospital operating rooms, then a resistance measuring instrument provided with a 1000-volt potential source should be employed in measuring floor resistance. This is especially true where high contact resistances exist, as they commonly do, between the electrodes and the floor.

Resistance values differing widely in magnitude have been obtained in measuring the resistance of floors in hospital operating rooms by using a 500-volt and 1000-volt instrument. Each of the two instruments was successively connected to the same pair of test electrodes which were not moved during the comparison tests.

With the application of a power supply of 1000 volts in the resistance-measuring instrument, the contact resistance between each electrode and floor surface may become decimated by virtue of harmless localized ionization discharge at the many points of light contact. These ionization phenomena occur in a like manner from electrostatic voltages of identical magnitude when impressed between any

floor-bearing facilities and floor.

Resistance - measuring instruments are provided with internal resistances which, in some instances, vary between wide limits. Unless the value of the internal resistance is known, a 500-volt or 1000-volt instrument, used in measuring a floor resistance whose value is commensurate with the internal resistance of the instrument, may actually apply across the floor resistance being measured only a small part of its rated voltage.

For example, one such 500-volt instrument possesses an internal resistance of 500,000 ohms; and, if the floor resistance being measured is about 500,000 ohms, the voltage applied to the test electrodes on the floor is only 250 volts rather than the rated 500 volts. On the other hand, if the floor resistance is 5 megohms, this same 500-volt instrument impresses a voltage across the two test electrodes of 454 volts, a value reasonably close to the 500-volt rating. Or, if the floor resistance is 100,000 ohms, this 500-volt instrument only impresses about 83 volts across the test electrodes on the floor—a voltage departing greatly from the 500-volt rating.

Several of the 500-volt and 1000-volt resistance-measuring instruments in common use possess internal resistances within the range of 80,000 to 100,000 ohms. But, on the other hand, some ohmmeters have internal resistances of several megohms. Floor resistances measured by these various instruments may vary widely by virtue of the differences in the relative voltages impressed across the test electrodes. Hence, the NFPA could well clarify this confusion by specifying, for its recommended upper and lower safe limits of floor resistance, the tolerance range of impressed voltages which are suitable in measuring floor resistances or by otherwise recommending suitable internal characteristics of ohmmeters.

The resistance between two test electrodes on a floor may reside largely in the contact resistances. When the measurement of the actual floor resistance is to be made in order to determine whether floors were initially constructed within the specified safe resistance limits, the test electrodes may then, and only then, be placed on spots of the floor which have been thoroughly cleaned by sandpapering. However, for all other measurements of floor resistance for the purpose of investigating whether the resistance of operating room floors, as normally maintained for day-to-day service, meet the specified safe resistance limits, the test electrodes should be placed about the floor area without spots being cleaned where the electrodes are located.

No water should be applied to the floor just prior to the resistance measurements; and, in fact, tests should only be conducted several hours after the floors have been washed—such as in the morning following the floor washing of the previous day, keeping a record of such pertinent information.

In order to provide a floor-bearing pressure for test electrodes more nearly commensurate with the values of floor-borne equipment in operating rooms, 5-pound electrodes equipped with three brass supporting legs were investigated. If the contact dimensions of each leg tip with the floor are ⅛″ by ⅛″, the pressure exerted on the floor by each leg tip is about 107 psi—a value more closely in keeping with those of the equipment utilized in operating rooms. The leg tips for these electrodes are used either bare or, preferably, with conductive paste applications—thus to reduce still further contact resistance. At this writing the author is not prepared to recommend the use of this measuring technique but, rather, he proposes that further study might well be devoted to

exploring its applicability.

Apropos of the suggested 3-leg 5-pound electrodes, as mentioned above, large-scale multi-legged electrodes were simulated in measuring floor resistance by using two conventional 16-pound metal stools resting on conductive linoleum flooring with two legs of one stool facing two legs of the other stool with a spacing of 3 feet between them. When the legs of each stool were provided with conductive rubber tips, the floor resistance between the two stools averaged 6.5 megohms; and, when the rubber tips on the legs were removed, the floor resistance between the two stools averaged 112,500 ohms. Later it is disclosed that the actual resistance of this 3-foot span of floor was about 5,000 ohms. This revealing fact, therefore, gives definite proof that practically all of the 6.5 megohms and, even, the 112,500 ohms were contact resistance.

These measurements are of significant importance in disclosing the dangerously high contact resistance provided by conductive rubber leg tips and casters on portable stools, tables, anesthetizing machines, and other such portable equipment in hospital operating rooms. This vast difference in measured values of resistance displays the vagaries in resistance measurements caused both by contact phenomena and by floor-bearing pressure effects.

Since the subject of measuring the resistance of flooring in hospital operating rooms is so important, safetywise, as well as in determining whether contractors have met standards of construction and terms of specifications in laying operating room floors, a program of research work is indicated for developing ways and means of providing really precise measurements of floor resistance, per se, rather than of contact resistances between the test electrodes and floor.

The author has devoted considerable study and test procedure to investigating resistance measurements on conductive linoleum in hospital operating rooms. Some of his data relating to resistance measurements on conductive linoleum between NFPA standard electrodes spaced from 6 inches to 12 feet apart are shown by the irregular curve, labelled A, in Figure 2. The conversion of this resistance curve to logarithmic ordinates is depicted by the straight line, labelled B, in the upper part of the chart.

With effective conductive paste applied to the bare metal faces of the NFPA test electrodes in their contact with the floor, the contact resistance when using the same ohmmeter is about halved. The improved resistance characteristic for the measurement of the same floor resistance is shown by the lower curve, labelled C, in Figure 2 for which the actual floor resistance is seen to be markedly reduced, homogeneous, and linear.

Thus, when the floor resistance of this conductive linoleum was measured in the manner suggested by the author, it is seen to be low and to possess a linear characteristic. On the other hand, when the floor resistance was measured by the standard NFPA method, the contact resistance between each electrode and floor is observed to be high and to possess a logarithmic rather than a linear function.

For this reason, in taking measurements by the NFPA method, the type of electrodes and their floor-bearing surface properties and pressure, the voltage applied to the electrodes by the ohmmeter, the surface condition of the floor, and the internal resistance of the ohmmeter, all are significant factors which influence the results.

In measuring floor resistance for any of the many purposes for which such data are used, the author suggests that tests be taken for several

linear distances and the data plotted as shown in Figure 2. By so doing, important information is disclosed regarding the resistance characteristics of the floor, the electrode contacts, and the ohmmeter behavior and, also, regarding the manner and care utilized in conducting the tests. Some such measuring technique, as herein used and explained by the author, is suggested for the consideration of NFPA.

Homogeneous Resistance for Floors

The homogeneity of flooring in hospital operating rooms is basically important in preserving everywhere on the floor-plane resistance values which lie within the recommended upper and lower safe limits. With moderately conductive floor coverings or plasticized coatings, continuity and homogeneity of the flooring are inherently provided, except possibly at over-laps or connective beading.

The NFPA disapproves the use in operating rooms of any flooring composed of discreet sections with their inevitable cracks for spawning infection, such as squares of rubber, asphalt, linoleum, cork or other compositions regardless of conductivity. Neither does it approve granolithic floors for the same reason, such as terrazzo, ceramic tile, and mosaic structures. In

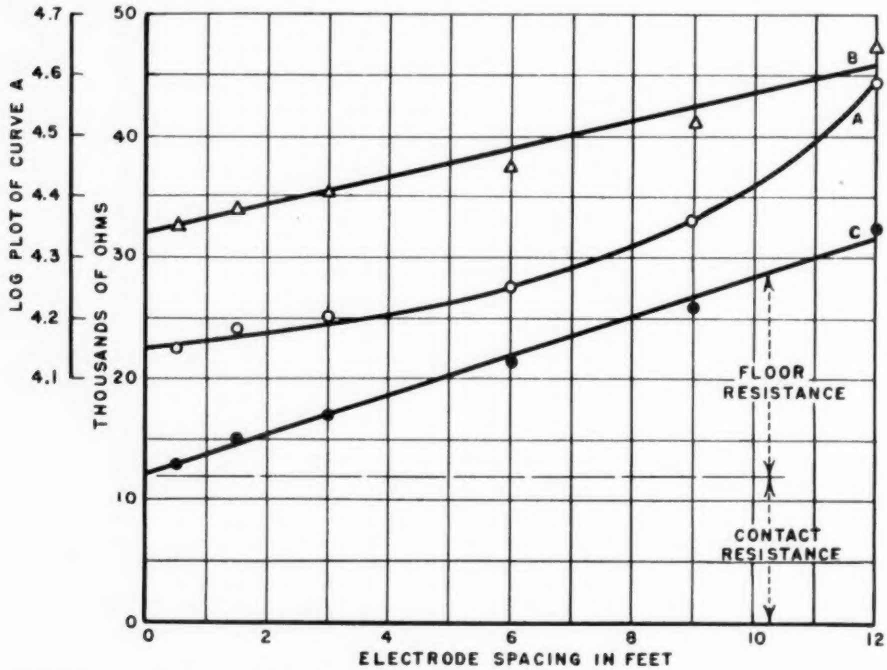

Figure 2. Curve A depicts the composite resistance characteristic of conductive linoleum flooring and of the electrode contacts, as measured by NFPA standard electrodes and a 500-volt d-c ohmmeter.

The linear log plot from the data of curve A, as shown by curve B, indicates the resistance characteristic to be a logarithmic function.

Curve C represents the resistance characteristic of the same floor, using the author's method of measurement described here, showing reduced and constant contact resistance and linear floor resistance.

such floors, the conductive cements, if used, wear away rapidly and leave high resistance flooring on which, thereafter, the floor-borne equipment rests.

Electrostatic Hazard Alarms for Instructing Operating-Room Personnel

After hospital operating rooms have been provided with satisfactory conductive flooring and with floor-borne facilities equipped with effective grounding devices, replacing all types of drag chains, to yield minimum contact resistance with floors, as well as personnel utilizing prescribed safety measures of wearing conductive clothing throughout and conductive footgear, the undertaking of rendering the operating room safe from electrostatic hazards has only, thus far, encompassed the physical phases of the safety conversion. At this stage enters the human element with its varied emotions, its errors of omission and commission, as well as its lack of adequate training in every phase of electrostatic hazards.

The laws of Nature are immutable and inexorable. Mother Nature is terribly unforgiving of any carelessness or neglect; and, she is most intolerant of ignorance or disregard in violating her physical laws. Hence, the provision of every known mechanical means for contributing to safety in operating rooms should be recognized at best as only physical aids in a comprehensive program which, also, must include education and training of operating room personnel for achieving safety in its fullest measure.

Starting with the premise that every known safety facility in the operating room has been properly and adequately provided to eliminate electrostatic sources of ignition, there follows a long, tedious, and meticulous educational and training program of safety which should be carefully conceived and vigorously conducted.

Certain techniques in operating room procedures which engender subtle and unsuspected creation of dangerous electrostatics require investigation and correction, such as, the flipping of sheets on or off operating tables or the sudden rising of a person from a metal stool. Experience, based on a well-organized educational program, will prescribe that, in order to reduce to nil these and other electrostatic hazards, such normally rapid actions as mentioned should be resolved to slow motion and specialized techniques developed for permitting time dissipation of electric charge and the consequential elimination of electrostatic sparks.

The instrumentality around which such an instructional program for operating-room personnel would be formulated comprises an electric field detecting instrument which, by being connected to an overhead antenna system in the operating room, detects and by an alarm gives evidence of electrostatic potentials in excess of a preset safety threshold at which spark and ignition hazards arise.

Since the threshold potential for creating sparks in operating rooms has been determined at about 1100 volts, this electric field detector should be adjusted to operate its alarm at a slightly less value—say about 1,000 volts. If adjusted for too low a voltage, the alarm would unnecessarily sound and repeatedly be "crying wolf." When this pre-set potential of 1000 volts is exceeded by cause of any one of many possibly ill-advised acts of operating-room personnel, the presence of such hazards is made known by a low buzz of the indicating instrument and by the lighting of a red signal lamp. Thus, in this manner, the operating-room personnel are alerted

to the fact that some of their manipulations or movements are causing hazards.

To illustrate, a person wearing rubber-soled shoes would find that the electrification at each step in the process of walking would, under favoring electrostatic conditions, sound the buzzer each time his shoe tips were successively raised from the floor. In addition to rubber-soled or composition-soled shoes, substantial leather-soled shoes commonly give rise to similar electrostatic impulses. Maximum safety, in this respect, prescribes the use of conductive footgear.

Such electric field detecting instrumentation, as described, in no wise eliminates or even affects the possibility of operating-room explosions. The system is wholly an instructional aid, and an important one, used to announce the occurrence of a hazardous electrostatic act by a member of the operating-room personnel. At the sounding of the alarm, an alert anesthetist would take instant cognizance of everyone's position in the room, what each was doing at the moment the alarm sounded, and following the operation or later, after purging the operating room of flammable vapors, would direct a re-enactment of the operating scene in an attempt to discover the source of the abnormal electrification sufficient to cause the alarm to operate.

Such explorative testing can be facilitated by the use of a portable electric field detector which may be located near each person, successively, while each repeats his acts as at the time when the alarm sounded. After the cause for the alarm has been determined, a study with detailed training should be instituted in order to eliminate any repetition of such hazards. Only in this progressive manner of persistent and determined effort can operating rooms be made safe, the lives of patients, nurses, and doctors protected, and the losses of physical property prevented.

The Bureau of the U. S. Naval Ammunition Depot at Crane, Indiana, prepared a brochure entitled "A REPORT — STATICATOR INSTALLATION"[4] which explains: the death-dealing hazards of electrification at this plant in the processing, handling, and bagging of gunpowder; the use of an electric field detector—called the Staticator—with systems of overhead antenna for discovering unsafe job practices; and, the instructional methods instituted to detect, isolate, and correct electrostatic hazards caused by plant operators.

The reading and study of this invaluable and interesting report of how a Staticator may be employed to resolve to complete safety electrostatic hazards, by detecting and correcting the causes of electrification and by installing a workable system of instruction for the personnel, is commended to each nurse, each anesthetist, each anesthesiologist, each surgeon, and each hospital administrator who is entrusted with the preservation of life and property during the specialized services performed in hospital operating rooms.

Obviously time, study, and perseverance are required to adapt each member of the operating room staff to his individual responsibilities in detecting and correcting, by a training program, the potentially hazardous conditions.

As a means of resolving theory into practice, an electrostatic field annunciator system was recently installed for trial in one of the major hospitals in the New York City Department of Hospitals. The objectives of this experimental installation embraced both salient phases of the educational ad-

4. Reprints of this report are available through the John Hewson Company, 106 Water St., New York 5. N.Y.

vantages offered by this system. These dual advantages are: 1—the detection and signalling of dangerous potentials as generated by inadvertent acts of operating-room personnel; and, 2—the subsequent isolation of the electrostatic causes which operated the alarm signals with the objective of resolving the apparent hazards and of providing group instructions, as a "dress rehearsal," to preclude repetition of the hazards.

A complete Staticator installation provides each operating room with an individual antenna, red light and buzzer alarms, and a control panel. In addition, a composite control panel to indicate alarms and signals from each of the operating-room antennae may be included in an installation and so located that it comes under the observation of the supervisor in charge of the operating rooms.

Training—
The Gateway to Safety

At least it should be reassuring to hospital operating room personnel to know that all electrostatic hazards, regardless of their origins or modes of expression, can be controlled; and, indeed, in hazardous areas of hospitals, all dangers from electrostatic fires and explosions may be effectively resolved by the diligent exercise of two concurrent approaches to the safety problem.

One approach to the safety problem lies in providing within hazardous areas all requisite physical agencies whose minimum standards are contained in the code of Recommended Safe Practice for Hospital Operating Rooms described in NFPA Bulletin No. 56. Essential safety provisions are: moderately conductive homogeneous flooring within the specified resistance range and maintained scrupulously clean day-by-day; anesthesia machines provided with conductive rubber parts throughout; the elimination of all grounding chains on portable equipment, using in their place, and without depending on grounding by highly erratic rubber casters and leg tips, a positive and resistance-controlled grounding device secured to each portable unit; and, various other physical facilities of safety, each being important and perhaps vital even though not specifically enumerated in this brief paper.

The other concurrent approach to safety relates to the human behavior of a specialized team of personnel, performing as it does its many and varied services within a hazardous area. The success of this phase of a comprehensive safety program within hospital operating rooms depends intimately and specifically on the individual knowledge, training, and responsibility of each member of the operating team. The safety achieved throughout the unified performance of the many duties of the team can be no more trustworthy than the acts of any single member. One thoughtless, precipitate, ill-considered action may jeopardize the safety of or, indeed, injure if not kill all members of the operating personnel, as well as the anesthetized patient. Safety is not a comparative term and how true is the widely advertised expression "you cannot be half safe."

Electrostatic hazards and their potentially dire consequences throughout anesthetizing areas must not be considered by hospital administrators an inevitable risk in the art of surgery by an operating team. Electrostatic problems in operating rooms arise wholly and only by man-made actions under certain variable conditions. Safety from the resulting fire and explosion dangers is the responsibility of the doctors, anesthetists, and nurses of the operating team to develop and acquire in their daily work, even as they

do in the other techniques practiced within their domain of service.

In developing and acquiring safety techniques from electrostatic dangers, adequate information is available in published form from which, by diligent application, the inquiring student may become self-instructed regarding the nature of static electricity, its generation, sparking potentials, ignition, dissipation, and explosion controls under hazardous conditions. Such a course of home study is indicated as a good start on the road to electrostatic safety for every surgeon, doctor, anesthetist, and nurse who serves on an operating team in surgery.

In addition, safety in the operating room from electrostatic hazards can be attained in practice through group courses of appropriate instruction and training. The anesthetist should be given the authority to acquire the inexpensive equipment necessary for demonstrating the properties and behavior of static electricity, its manner of igniting flammable mixtures, and the advantages of various control methods for rendering conditions safe in operating rooms. Then a regularly scheduled course of instruction with demonstrations could be given by the anesthetist to all who have a part in surgery. In such instruction, the Staticator should serve as the focal center of all demonstrations, using it as a means of detecting hazardous electrification and of showing how safety may be attained.

When instruction of this character is included as a requisite course for medical degrees and for graduate nurses and anesthetists, a significant advance will have been made toward really promoting safety from electrostatic hazards in operating rooms. In this manner, the education and training of doctors and nurses in this important area of safety will be based on an enduring, successful, and worthy program of continuing instruction.

The long outmoded "pick-up" method of learning, with its infected harvest of notorious faults, continues in vogue throughout hospitals and it is still tolerated by doctors, anesthetists, and nurses who are trying to acquire by the hard way the essential techniques of controlling electrostatic explosion hazards. Doctors would be horrified, and rightfully so, should the discredited "pick-up" method of learning in this day and age be applied to educating and training in the science and technology of their profession.

Safety in operating rooms is of serious concern and a vital matter to the hospitalized public who, in undergoing surgical administrations, have the expectation of normal recovery from their bodily ills rather than the more grievous consequences of falling prey to inexcusable derelictions of professional services which inflict other brands of injuries or even death. Hence, in the public interest, as well as the welfare of hospital personnel, safety principles and practices relating to electrostatic explosion hazards, as they apply to the day-by-day performance of surgical teams in operating rooms, should be inculcated on the doctors, anesthetists, and nurses by the most modern instructional methods and through the best informational channels currently available.

Until the hazardous areas within hospitals are rendered safe from explosions and their frightful consequences by instituting corrective techniques through education and training, the finger of public censure will be pointed with unwavering and critical reproach directly at hospital, medical, nurse and anesthetist associations for their collective negligence, complacency, and tolerant attitude toward this spreading stain of defeatism on their escutcheons of professional responsibility.

The Influence of Body Posture upon Arterial and Venous Blood Pressure in Gynecologic Surgery

C. Paul Hodgkinson, M.D., and Robert C. Rood, M.D.*
Detroit

The influence of body posture upon the hemodynamics of circulation is appreciated but not always applied to everyday surgical problems. Manipulation of body position as therapy is standard practice. The head-down position for acute arterial hypotension is commonly referred to as the shock position. Shock blocks for elevating the foot of the bed or stretcher are commonly found in hospitals.

Less well recognized as a therapeutic measure is the head-elevated position. The relief afforded the patient with cardiac disease by the head-up position attests to its favorable influence upon circulatory hemodynamics for patients with impaired cardiac function.

Precise definition of indications for the use of position therapy in surgical patients is generally lacking. How alterations in body position affect the circulatory system of anesthetized patients is seldom given serious consideration. As a result, anesthetized patients are tilted in all directions indiscriminately as the need for surgical exposure arises.

Clinical experience with gynecologic patients suggested that alterations in body position during operations are more important than is generally appreciated. Too frequently after an uneventful operation, acute circulatory failure, characterized by acute arterial hypotension, has been observed. That this state is not incident to blood loss is suggested from the time of occurrence of the vascular collapse. Blood lost during surgery is replaced promptly, and this prevents acute arterial hypotension during operations.

The acute hypotension usually observed after gynecologic procedures does not appear to be related to blood depletion. It occurs after termination of the operation, frequently after the patient has been returned to bed. Examination discloses a low or unobtainable blood pressure, cold extremities, and a pulse of poor quality. So inconsistent is this shocklike state with the estimated blood loss and the magnitude of the operation that one is at a loss logically to explain this sudden deterioration of the patient's condition. Elevation of the foot of the bed, administration of fluids intravenously, and administration of drugs to stimulate the vasomotor system usually promptly relieve the condition.

This investigation was undertaken to study the effects of alteration in

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body position upon the hemodynamics of gynecologic patients. Patients in good general condition and of comparable physical status subjected to hysterectomy by one gynecologist were used for this study. General anesthesia with ethylene, cyclopropane, and at times a small amount of ether was employed. At times *d*-tubocurarine chloride was used as a muscle relaxant.

To study the influence of alteration of body position upon venous return, blood pressure in the ovarian vein was measured with patients in three positions[1]. Using the method of Von Tabora the ovarian vein pressure was measured with the patient in the horizontal position, with 15 degrees reverse Trendelenburg, and with 15 and 20 degrees Trendelenburg.

Because there are no valves in the central veins, such measurements give information relative to the effects of gravity upon the central venous pressure. With the patient in the horizontal position, the average venous pressure recorded was 14.82 cm. water. Tilting the operating table head-upwards 15 degrees (reverse Trendelenburg) resulted in an increase of the average venous pressure to 19.66 cm. water. Tilting the operating table head-downwards 15 degrees (Trendelenburg) shifted the weight of the blood away from the point of measurement in the ovarian vein because the average venous pressure decreased to 8.18 cm. water pressure.

These studies indicated that gravity considerably affected the central venous pressure. The pressure could be expected to be high if the column of blood above the point of measurement was increased. Tilting the patient to the Trendelenburg position shifted the greatest pressure in the central veins to the level of the heart. Through gravity the Trendelenburg position caused the blood pooled in the lower half of the body, including the legs, to flow into the central veins and the heart.

It appeared reasonable to assume that if return circulation was aided through tilting the operating table 15 degrees head-downward, an additional tilt of 5 degrees would bring about additional benefit. Much to our surprise, the average ovarian vein pressure with the patients tilted into 20 degrees Trendelenburg was increased rather than decreased. In the 20 degree tilt the average ovarian vein pressure increased to 10.11 cm. water. This increase in ovarian vein pressure mainly occurred in two patients who were suffering from disturbances of cardiac function. One patient had a large cartoid aneurysm. She demonstrated no distress when placed in the 15 degree Trendelenburg position. However, with the table placed in the 20 degree position, the patient became distressed. The ovarian vein pressure rose to 10 cm. water pressure from a previously recorded 4 cm. in the 15 degree position. The anesthetist rightly refused to leave the patient in this position. When the operating table was returned to the horizontal position, the condition of the patient improved, and the operation was completed with the patient in this position. Another patient suffering from severe arteriosclerosis and auricular fibrillation demonstrated a similar reaction. We also noted that obese patients were prone to show this paradoxical rise in ovarian vein pressure.

It was our impression that these paradoxical ovarian vein pressure reactions to the 20 degree Trendelenburg position were indications of distress due to central reflexes. Sudden overburdening of the heart with increased blood volume in the large

1. Hodgkinson, C. P.: Ovarian vein pressure studies. Am. J. Obst. & Gynec. 61:321-329, Feb. 1951.

veins was in most instances well tolerated. Patients with impaired cardiac function were distressed by this additional cardiac burden. Similar reactions are known to occur with administration of large volumes of fluid to the patient with cardiac disease or asthma and the obese patient.

In the study of arterial blood pressure in relation to alterations in body position a related, but somewhat opposite, physiologic principle was involved. Three groups of patients were studied. All patients were operated upon in the 15 degree Trendelenburg position. Upon termination of the operation, patients in group I were maintained in the Trendelenburg position until they had recovered from the effects of the anesthesia. Patients in group II were abruptly placed in the horizontal position. Patients in group III were removed from the operating table to a stretcher with 15 degrees reverse Trendelenburg.

Group I.—The arterial blood pressure in this group of patients demonstrated little variation from that recorded during the operation. None of the patients demonstrated acute hypotension, and in no instance was it necessary to administer fluids or drugs to stimulate the vasomotor system. Gradual return to the level position as the patient reacted from the effects of the anesthesia resulted in no adverse effects in the arterial blood pressure.

Group II.—The average postoperative arterial blood pressure reaction of this group was less favorable. Two patients of the 10 studied demonstrated depression in the arterial blood pressure and required supportive treatment. Each patient was given a transfusion of 500 cc. blood and vasomotor stimulants.

Group III.—This group of patients was abruptly removed from the 15 degree Trendelenburg position to a stretcher slanted head-upwards 15 degrees. At the termination of the operation the average systolic blood pressure was 128 mm. Hg. and the diastolic pressure 65 mm. Hg. Ten minutes in this position resulted in a decrease in the average arterial blood pressure. The systolic pressure fell to an average of 68 mm. Hg. and the diastolic pressure to 46 mm. Hg. In one patient the arterial blood pressure was unobtainable. In all except one instance it was necessary to return the patient to the Trendelenburg position. Vasomotor stimulants were necessary for three patients, and the other two patients were given blood transfusions. All recovered uneventfully.

Analysis of the physiologic adjustments to the Trendelenburg position suggested that the head-downward position mobilized the blood pooled in the lower half of the body to the general circulation. This increased available blood volume adequately compensated for the blood loss incident to the operation and to the diminished vasomotor control resulting from general anesthesia. Abrupt shifting of a patient from the Trendelenburg position to a head-upwards position resulted in a sudden shift of the blood volume to fill the veins of the lower half of the body causing depletion of blood volume from the general circulation and the production of hypotension.

Discussion

Studies by others have shown that cardiac output, arterial blood pressure, and pulse rate vary as the result of alterations in body position in normal subjects. Brigden, Howarth and Sharpey-Schafer[2] studied normal sub-

2. Brigden, W.; Howarth, S., and Sharpey-Schafer, E. P.: Postural changes in the peripheral blood-flow of normal subjects with observations on vasovagal fainting reactions as a result of tilting, the lordotic posture, pregnancy, and spinal anesthesia. Clin. Sc. 9:79-91 May 1950.

jects as they react to passive tilt. The head-up position resulted in decreased right auricular pressure and a diminution in cardiac output. When normal subjects were abruptly tilted head-up, 8 to 10 per cent fainted. It has been postulated that such reactions occurred because of inadequately functioning circulatory reflex mechanisms.

Hypotension has been noted frequently in obstetric patients. Howard, Goodson and Mengert[3] studied this problem and found that 10 per cent of women who lie recumbent for any length of time during late pregnancy develop hypotension within five to ten minutes. They noted that these patients at times demonstrate a shock-like syndrome except that they fail to develop tachycardia. Vast changes occur in the circulation during pregnancy. The relative distribution of blood is greatly increased in the lower half of the body. Venous pressure in the veins of the legs is tripled in late pregnancy[4]. The ovarian vein pressure in the horizontal position remains essentially unchanged and averages 15.43 cm. water[5]. Some idea of the redistribution of blood can be gained through study of the ovarian veins during pregnancy. The caliber of the ovarian vascular pedicle increased 2.86 times by term, and the estimated increase in blood volume carried by the ovarian veins is over 66 times. These studies suggest that alterations in body position are of increased importance in pregnancy.

Depressant drugs interfere with cardiovascular physiology. Drew, Dripps and Comroe[6] compared the reaction to tilting before and after the administration of morphine. Twenty-five subjects were studied. Before the administration of 0.20 mg. morphine, two of the patients fainted when they were tilted to the 75 degree head-upwards position. After morphine 11 of the same 25 subjects fainted. King, Elder and Dripps[7] studied patients given meperidine and found a similar but less severe reaction.

It is logical to expect the same reaction from general anesthetics. Peterson, Eather and Dripps[8] noted that 14 of 31 patients operated upon in the lithotomy position showed a decrease in arterial blood pressure when they were returned to the horizontal position. The decrease in systolic arterial blood pressure ranged from 25 to 60 mm. Hg. Cole[9] studied the head-lowering treatment for hypotension following surgical operations. His study emphasized the effects of gravity after tilting. Tilting a supine patient head-downward resulted in a decrease in arterial pressure in the legs and an increase in arterial pressure in the arms. Cole postulated that the head-down position favored circulation to the vital organs.

The present investigation supports the work of others. In hypotension the head-down position favors mobilization of the blood filling the veins of

3. Howard, B. K.; Goodsen, J. H., and Mengert, W. F.: Supine hypotensive syndrome of late pregnancy. Obst. & Gynec., to be published. Address given at 1st Clinical Session, Am. Acad. Obst. & Gynec., Chicago, Dec. 1952.

4. McLennan, C. E.: Antecubital and femoral venous pressure in normal and toxemic pregnancy. Am. J. Obst. & Gynec. **45**:568-591, April 1943.

5. Hodgkinson, C. P.: Physiology of the ovarian veins during pregnancy. Obst. & Gynec. **1**:26-37, Jan. 1953.

6. Drew, J. H.; Dripps, R. D., and Comroe, J. H.: Clinical studies on morphine. II. The effect of morphine upon the circulation of man and upon the circulation and respiratory responses to tilting. Anesthesiology **7**:44-61, Jan. 1946.

7. King, B. D.; Elder, J. D., Jr., and Dripps, R. D.: The effect of the intravenous administration of meperidine upon the circulation of man and upon the circulatory responses to tilt. Surg., Gynec. & Obst. **94**:591-597, May 1952.

8. Peterson, L. H.; Eather, K. F., and Dripps, R. D.: Postural changes in the circulation of surgical patients as studied by a new method for recording the arterial blood pressure and pressure pulse. Ann. Surg. **131**:23-30, Jan. 1950.

9. Cole, F.: Head lowering in treatment of hypotension. J.A.M.A. **150**:273-274, Sept. 27, 1952.

the lower extremities. Abrupt elevation to the head-up position suddenly depletes the heart and its large vessels of a large volume of available circulating blood. As a result of anesthesia the vasomotor reflex compensatory control to the vascular bed is lost. Although the major effect is on the arteries, the veins also suffer. With such control lost the venous system passively responds to the effects of gravity.

Circulatory collapse occurs when the effective blood volume is diminished. Asmussen, Christensen and Nielsen[10] have estimated that normally 550 cc. of blood can be pooled in the legs. Placing a patient in the Trendelenburg position mobilizes this blood to the general circulation. During gynecologic operations this augmented blood volume probably compensates for the blood lost. Buchman[11] studied the blood lost during gynecologic operations and found that the average hysterectomy resulted in the loss of 678 cc. Evidently, during the surgical procedure of hysterectomy with a patient in the Trendelenburg position, the blood lost is adequately compensated for by the position of the patient.

Upon termination of the operation the tilting of the patient abruptly upwards from the Trendelenburg position subjects her to a rather sudden depletion of effective blood volume. In the absence of adequate vasomotor control, with the sudden withdrawal of the combined blood lost and the amount required to fill the veins of the legs, vasomotor collapse is to be expected. If the foregoing figures were used, this would amount to 1228 cc. blood lost from the circulation. It appears unfair to subject a patient to anesthesia, remove her uterus, lose a large volume of her blood, and then suddenly place her in a position that further depletes her effective circulating blood volume.

Our studies have suggested, on the other hand, that placing a patient in the Trendelenburg position may embarrass her cardiac function. This is especially true of patients suffering from asthma, obesity, or impaired cardiac action. In addition to pressure on the diaphragm from the abdominal viscera, sudden augmentation of the circulating blood volume may be a factor of importance. Cardiologists have constantly warned against administering large volumes of fluid intravenously to patients with cardiac disease for fear of overloading the heart. The head-down position, in effect, does just that.

Management

The management of patients operated upon in the Trendelenburg and the lithotomy position requires consideration of the altered hemodynamics of circulation effected through changes in body position. Appreciation of the depressing effect upon blood vessel physiology and the increased significance of gravity as it affects the distribution of circulating blood volume is essential. Whereas impaired reflex control can probably make adjustments to the effects of gravity if alterations in position occur gradually, abrupt changes in body position should be avoided. This warning applies to both abruptly placing a patient in the Trendelenburg position and removing her from it. Gynecologists should heed the apprehension of anesthetists if distress is evident when a patient is tilted into the Trendelenburg position.

Recognition that the immediate postoperative recovery period is a phase best handled by anesthesia per-

10. Asmussen, E.; Christensen, E. H., and Nielsen, M.: The regulation of circulation in different postures. Surgery **8**:604-616, Oct. 1940.

11. Buchman, M. I.: Blood loss during gynecological operations, Am. J. Obst. & Gynec. **65**:53-64, Jan. 1953.

sonnel has been an important safety measure. The designation of space for postanesthesia recovery rooms is a modern hospital contribution. Recovery rooms should be staffed with personnel conscious of the unique requirements. The physical equipment should be adequate. Facilities to elevate the foot of the stretcher or bed should be available. Shock blocks that require the brute strength of a strong orderly, a physician, or nurse have been antiquated by mechanical devices. At Henry Ford Hospital Mr. Krolicki of the Department of Maintenance constructed a hydraulic bed jack that can be operated by the most frail nurse. This bed jack is operated after the fashion of an automobile jack and has been of distinct benefit to patients postoperatively. Another therapeutic measure of proved value is wrapping the legs from toes to thighs with elastic bandage before removing the patient from the Trendelenburg position. This procedure prevents pooling of blood in the leg veins and thereby aids in maintaining the available circulating blood volume. Facilities for the administration of oxygen, fluids intravenously, and vasopressor drugs are necessary.

Anesthesia for Orthopedic Surgery

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Indianapolis

The requirements for anesthesia for orthopedic surgery vary from the simple infiltration of the hematoma in the case of a Colles' fracture to intratracheal intubation for the patient to be operated on in the prone position. The short procedure of reducing a simple fracture about the wrist or ankle may require little consultation between the anesthetist and the surgeon, but the needs of the prolonged anesthesia for spinal fusion for scoliosis should be discussed before the operation is started.

Anesthesia for orthopedic procedures may be divided into two large groups, that is, anesthesia for the emergency case and anesthesia for the elective case.

Emergency Surgery

Patients for emergency operations may be classified into two groups, those who require short procedures, and those who, owing to the nature of the injury, require prolonged anesthesia.

There is no such thing as minor anesthesia for minor injuries. The child who fractures both bones of the forearm and is admitted to the hospital soon after a meal, may vomit and aspirate the food with grave complications. It is far better to immobilize the arm without anesthesia and postpone the reduction until proper preoperative preparations can be made. General anesthesia may then be administered with relative freedom from danger. In such a case we prefer to give atropine in the indicated dosage about one hour before the anesthesia is started. Ether is the safest anesthetic. The use of oxygen and carbon dioxide during induction will usually result in a smooth induction, and recovery from the anesthesia will be uneventful. Vinethene may be used for the induction, but it must be used with caution. We believe it has no advantage over ether in the hands of an experienced anesthetist. Vinethene, furthermore, must not be used for prolonged procedures, such as a difficult reduction. The onset of twitching of skeletal muscles does not call for more of the agent; it is the signal for immediately discontinuing the administration and switching to ether, with care being taken to keep the airway patent and the supply of oxygen adequate.

The patient with a Colles' fracture should present no great problem from the standpoint of anesthesia. Local infiltration with 1 per cent novocain will give satisfactory anesthesia in almost all instances. Nitrous oxide-oxygen anesthesia might be satisfac-

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tory. No one recommends a concentration of nitrous oxide of over 80 per cent. Usually a 75 per cent nitrous oxide-25 per cent oxygen mixture proves satisfactory for these short procedures. Recovery is rapid, and the patient is soon able to go home. Pentothal sodium anesthesia is attended by a prolonged recovery time, and this is an inconvenience. A combination of the two agents might be used for this type of procedure with a recovery time slightly longer than that with nitrous oxide-oxygen alone, but shorter than that with pentothal sodium alone. Emesis rarely occurs.

Premedication for a simple procedure must not be overlooked. According to the age and size of the patient, morphine and atropine should be administered at least a half an hour before starting the anesthetic. The technic of giving the premedication is important. Intravenous administration is sometimes useful in this respect.

The severe orthopedic emergency, such as the extensive open fracture of one of the long bones, requires judgment. In almost all cases sufficient time may be taken to prepare the patient for anesthesia. If the patient is in shock, administration of blood and plasma may be indicated, and other therapeutic measures may be necessary to prepare the patient for the anesthesia and surgery. Open drop ether anesthesia is probably the safest choice. although the combination of pentothal sodium intravenously and nitrous oxide-oxygen is usually safe in the hands of an experienced anesthetist. Spinal anesthesia should not be used if the patient has suffered shock. Nitrous oxide should never be used to the point of anoxia, because of the hazards of cortical damage with temporary or permanent mental and emotional changes. Sudden death may occur on the table if the oxygen intake is not sufficient at all times.

The induction of anesthesia in the case of a child deserves special consideration. Children are usually frightened, and the anesthetist may accomplish a great deal in allaying the fear. Offering reassurance rather than holding the patient is the ideàl approach to the induction. The child may be told that he is going to sleep and that the mask will not hurt. If vinethene or ether is used, the mask should be brought to the face slowly while the child is urged to breathe deeply. Oxygen and carbon dioxide often make induction easy by producing hyperpnea. If the child cannot be placated and will not respond, there is nothing to do but proceed. It is unwise to administer oxygen or carbon dioxide to a crying, struggling child. If he is given oxygen, he may have acapnia, which delays induction. An excess of carbon dioxide may produce distressing convulsions that might confuse the anesthetist. Properly, the child should be put to sleep promptly without delay. In the case of an inpatient, the administration of pentothal sodium by rectum preoperatively is certainly an approach to this problem. For older children pentothal sodium intravenously combined with nitrous oxide-oxygen has given us satisfactory results. At no time should anoxia be tolerated.

Elective Surgery

Fortunately, many surgical procedures in orthopedic surgery are elective. The general condition of the patient, age, state of nutrition, blood pressure, blood picture, kidney function, and over-all physical capacity may be evaluated before operation.

The preoperative medication is of great importance. It should be given long enough before induction to have taken effect. The actual administration is the responsibility of the ward

nurse. The medication prescribed is chosen according to the age, condition, and susceptibility of the patient. The manner of administering the drug is important. An injection into the adipose tissue of an obese person will require a much longer time for absorption than an injection into the muscle of a lean person. Many anesthetists prefer scopolamine to atropine because scopolamine is a weaker vagus nerve depressor. This may be important in nitrous oxide-oxygen anesthesia, because with atropine premedication the concentration of oxygen may be low enough to produce anoxia. Since scopolamine is depressing, the amount of morphine given with it should be about half the usual dosage. Susceptibility to morphine should be determined before administration. Morphine should be used with the greatest caution for old patients and the very young. Morphine should be given at least one hour before induction of anesthesia so that the depressing effects of the drug will in great part have passed away. If barbiturates are used, the dosage should produce drowsiness rather than deep sleep, because the effect of the drug may mask early signs of asphyxia that later may develop into anoxia. Anesthesia is usually the easiest to induce in the adequately premedicated patient.

The anesthetic agent to be used will depend on the type of operation to be performed, the part of the body involved, and the position of the patient during the operation. For operations on the lower extremities there is a wide choice of anesthetizing agents. Spinal anesthesia is a most useful method. Of course, the contraindications for its use may not be disregarded because of its convenience. By positioning the patient and the use of the factor of specific gravity of the spinal anesthetic, it is possible to limit the anesthetized area. This will reduce the hazards of hypotension. It is possible to prolong the effect of spinal anesthesia by the addition of vasoconstricting drugs to the anesthetic solution or by the use of pontocaine or other accepted long-acting agents. The lowering of the blood pressure may be counteracted by using vasoconstricting drugs. Nausea and vomiting may be prevented with intravenous administration of pentothal sodium in small dosage, just enough to produce drowsiness for a short period. Nausea with vomiting is usually of short duration. We have had a very low incidence of so-called spinal headaches. The cause of these headaches is unknown. It is stated that elderly patients do not tolerate spinal anesthesia because of the hypotension produced. We have operated on many elderly patients under spinal anesthesia and to date have not encountered a major complication. Spinal anesthesia is contraindicated for children.

Ether has been and remains the safest anesthetic agent. It can be given without any or few of the bad effects. With ether anesthesia a seriously ill patient can be carried through a major surgical procedure if it is correctly administered and the patient is properly prepared. The notorious bad effects of ether are not caused by ether but are due to anoxia from the anesthesia produced. Oxygen may be given with ether whenever indicated. Many patients are reacting from the anesthesia before leaving the operating room. The anesthetist's timing in relation to depth of anesthesia is of great importance in the early recovery of the patient. We prefer ether to any other inhalation anesthetic for young or elderly patients.

As a rule, surgical procedures on the upper extremity require general anesthesia. Ether remains the best and safest agent. For the average adult

patient pentothal sodium-nitrous oxide-oxygen is satisfactory. We do not like the combination for children because the amount of pentothal sodium needed is too great. In the elderly patient the effects of pentothal sodium are too lasting.

Operations on the spinal column require the best judgment on the part of the anesthetist and surgeon. The prone position is compromising to gaseous exchange. Here anoxia may be very difficult to combat or prevent. The positioning of the patient on a properly designed frame or the placing of sand bags under the iliac crest will prevent or diminish the difficulties.

We prefer ether anesthesia to all other types of general anesthesia for operations on the spine. Intubation is desirable but not absolutely necessary. However, intratracheal intubation should make the surgeon feel secure in the thought that positive control of the oxygen intake is in effect. With the patient in the prone position anoxia is the greatest hazard, and the anesthetist must be constantly diligent in its prevention. This cannot be overemphasized.

Call to the Convention

As provided for in the Bylaws of this association, and at the direction of Mrs. Josephine B. Bunch, president, we hereby issue this official call to the members of the annual meeting to be held in San Francisco, August 31-September 3, 1953, at the Civic Auditorium. The annual business session will be held on Tuesday, September 2.

Accomplished at the Executive Offices, 116 S. Michigan Ave., Chicago 3, Ill., this 20th day of July 1953.

(Signed) FLORENCE A. MCQUILLEN, R.N.
Executive Director

Trichorethylene

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The purpose of this article is to discuss an anesthetic agent that is relatively new in this country with respect to its application in surgery and obstetrics.

History

Trichlorethylene was discovered in 1800 in England, and now it is rapidly gaining favor in the United States. In England trichlorethylene as an inhalation agent was reported and recommended in 1941 by Hewer, although its use in the United States had been reported in 1934. Revival of interest in trichlorethylene as an agent for inhalation anesthesia was prompted by the quest for a nonflammable potent inhalation anesthetic agent. After Hewer's authoritative report, its employment in Great Britain spread widely and rapidly. Its popularity in the United States has grown with amazing speed, and today it is used in many fields of anesthesia.

Physical and Chemical Properties

Trichlorethylene, $CHCl:CCl_2$, is a colorless liquid with a sweet pleasant odor. The molecular weight is 132.5; the specific gravity is 1.47 at 15.5 C.; the boiling point is 87 C. It is practically insoluble in water, but it is miscible with chloroform and ether. Trichlorethylene is colored with a bluish tint to distinguish it from chloroform. Since the boiling point of this agent is 87 C., it cannot be vaporized adequately; therefore it is necessary to employ a special vaporizing apparatus.

It is important that the preparation be pure and suitable for use by inhalation. It should be stored in the dark and away from air and sunlight. As prepared for its many commercial uses it contains impurities and degenerating products. So only specially prepared trichlorethylene should be used. It has been assumed that it is the ideal analgesic; so far there is no other drug with which analgesia can be produced so easily, so quickly, and so safely with less expense.

Trichlorethylene may be heated without decomposition, but if its vapor is diluted with air and is exposed to an open flame, decomposition will occur, giving rise to dichloracetylene and traces of phosgene, which is a very poisonous gas. It cannot be used in a closed soda lime rebreathing system. It is important to remember that trichlorethylene must never be allowed to pass over soda lime, as this will possibly cause decomposition of the product with production of potentially toxic substances.

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Effects on Various Organs

Trichlorethylene causes less liver damage than ether. Renal function is not altered to any noticeable degree. The metabolic rate appears to be very little disturbed. The effect of the agent on the cardiovascular system is still controversial and does not seem to be a serious factor. Vagal tone seems to be increased thereby inducing bradycardia if the rate of anesthesia is pushed. It has no effect on coronary vessels. There is no significant deviation in blood pressure. The pupils do not dilate in deep anesthesia.

The effect upon the central nervous system is usually analgesia without unconsciousness and without untoward reactions. Overdosage is generally followed by numbness. When administration is discontinued, recovery is rapid. In the usual dosage trichlorethylene does not affect the respiratory system. No excessive salivation or mucus occurs, and nausea and vomiting are infrequent.

Effects on Respiration

Trichlorethylene has no irritating effect on the respiratory passages, and it does not complicate or contraindicate the use of any other agent. The reason for the increased rate of respiration has been the subject of much work, and a reasonable explanation is as follows:

The Hering-Breuer reflex consists of two parts, (1) expansion of the chest inhibiting further activity of the respiratory muscles and (2) collapse of the chest initiating respiratory efforts. Until recently it was supposed that the whole reflex could be explained on the basis of one set of afferent or stretch fibers from the pulmonary alveoli up the vagi to the respiratory center. It now seems probable that a second set of fibers, running from the small blood vessels in the lungs to the vagi, are responsible for the deflation reflex. From animal experiments it appears that all the volatile anesthetics sensitize the stretch reflexes and can thus cause shallow respiration. The deflation reflexes, on the other hand, are affected differently. For example, ether first stimulates and then paralyzes them, while trichlorethylene causes stimulation throughout, so that in deep narcosis the respiratory rate increases with the concentration of the drug.

Techniques of Administration

Trichlorethylene is a highly potent analgesic agent, but it does not readily induce muscular relaxation. The vapor concentration for relief of pain is well below the toxic level. The amount necessary to produce surgical anesthesia is likely to cause arrhythmias; therefore it is not recommended for surgical anesthesia.

Self administration is not recommended for children under 2 years of age, but there is no age limit for older patients requiring analgesia.

Trichlorethylene makes possible a smooth induction in a short time, but as a single agent it does not produce profound muscular relaxation. It has been found that once muscular relaxation is established with the addition of ether, this relaxation can be maintained for a fairly long time by use of a separate attachment on the gas machine.

The patient should be psychologically prepared and assured that the procedure will be a pleasant one without pain and with a rapid recovery.

There are three types of trichlorethylene inhalers on the market: the Cyprane Inhaler, the Duke Inhaler, and the Airlene. Trichlorethylene has been used with all three types of inhalers at St. John's Hospital for over 1,000 cases. Trichlorethylene will re-

act with the rubber parts of an anesthesia machine, resulting in their decomposition.

Uses of Trichlorethylene

Trichlorethylene has been used effectively in obstetrics, in surgery, in the emergency room, and in the patient's room.

As an analgesic agent it is valuable for minor operations. Special uses are in treating burns and changing painful dressings, for the reduction of fractures, incision and drainage of abscesses, and suturing of superficial wounds. It has been found useful in pediatrics, both as an analgesic and as an anesthetic.

Trichlorethylene in Obstetrics

In obstetrics trichlorethylene is a safe and an efficacious method of analgesia and amnesia. It has a very slight effect, if any, on the uterine musculature; therefore if uterine relaxation is needed, it is necessary to change to another agent.

Amnesia occurs in nearly all cases of anesthesia with trichlorethylene whether it is used alone or in combination with other agents. With intermittent use of the trichlorethylene inhaler, the first stage of labor can be controlled so that it is not necessary to use any barbiturates.

There are no depressing effects on the baby, and the patient's attitude is most gratifying. The inhalers are so constructed that the concentration can be adjusted to the needs of the particular patient. The intermittent administration is continued with pains until time of delivery, the concentration being increased and continued until the shoulders are delivered. Trichlorethylene may be used for all uncomplicated deliveries.

Trichlorethylene has no adverse effect on the strength of uterine contraction and does not prolong the course of labor. With self-administered trichlorethylene analgesia, normal contractions are maintained and the patient is not only free from pain but is conscious and able to co-operate by bearing down. In the maternal blood oxygen level there is no demonstrable reduction in oxygen intake. The danger of fetal anoxia is negligible.

The technic of administration of trichlorethylene for obstetrics is as follows: The inhaler is filled with trichlorethylene (15 cc.) and strapped to the patient's wrist. As soon as the patient feels a pain, the mask is placed over the face and the patient is instructed to take several deep breaths, continuing as long as she feels pain. If at first the odor is disagreeable, the mask is lifted and again reapplied as needed.

Trichlorethylene in Dentistry

Indiscriminate use of trichlorethylene for analgesia is unwarranted as is the indiscriminate use of any other drug. Analgesia eliminates many discomforts that accompany dental procedures, i. e., the drilling of teeth for cavity preparations and innumerable other dental procedures that may be painful or extremely uncomfortable.

Analgesia is a useful adjunct when local anesthesia alone is not sufficient for operative dentistry. Routine extractions started with trichlorethylene analgesia may be followed by a completely painless injection. In such cases, after the injection, maintenance analgesia can be particularly valuable in lengthy surgical procedures using the nasal unit.

All equipment should be in readiness for immediate use. This includes the analgesic vaporizer, mouth props, saliva ejector, and all instruments to

be used for the completion of the procedure. The position of the patient in the dental chair is of the utmost importance. The nosepiece should be properly applied and the mouth props properly adjusted. The exhaling valve on the nosepiece is always open for the elimination of carbon dioxide.

Reactions to trichlorethylene vary greatly in different patients. Therefore there is no set percentage mixture to use as a guide in administering the drug. Inhalations and exhalations are carefully noted and the analgesic stage is maintained. It is suggested that no epinephrine be used with a local anesthetic and trichlorethylene because it may cause cardiac irregularities. The dental patient may be kept in a very light stage of analgesia and is capable of carrying out simple instructions during the procedure.

Trichlorethylene in Pediatrics

Trichlorethylene produces satisfactory analgesia for various minor diagnostic procedures in pediatrics, such as venipuncture, lumbar puncture, marrow puncture, abdominal palpation, and chest exploration, which cause apprehension and discomfort, often sufficient to render a child unco-operative or even actively resistant.

For instance, a child who is highly emotionally upset by a visit to the physician's office for an injection readily inhales a small amount of trichlorethylene, and while he is in an analgesic state, the injection can be painlessly carried out.

Trichlorethylene for Emergencies

When trichlorethylene is used, débridement of minor lacerations in the emergency room may be done with greater efficiency as well as comfort to the patient, as all burns require a thorough cleansing and débridement, and it may be attended to immediately upon admission. It is of the utmost importance to perform débridement and cover the raw surfaces as soon as possible.

Shaving of redundant granulations in old burns has also been simplified by the use of trichlorethylene analgesia, combined with the use of the Brown Electro Dermatome, and has resulted in a smooth surface ready for immediate grafting. Trichlorethylene does not cause a change in blood pressure, and oozing from the wound is noticeably less than when other anesthetic agents are used.

Shock

There has been no evidence of toxicity, and there appears to be no damage or shock from administration of trichlorethylene for long periods. The combination of trichlorethylene with ether and nitrous oxide and large percentages of oxygen added to the mixture may prove more suitable than any other general anesthetic.

Premedication

Although premedication is of sedative value, it is not necessary with trichlorethylene, as it does not stimulate the production of salivary secretions. The patient awakens completely within two to five minutes of the time the mask is removed.

Antidotes

There are no precise antidotes for trichlorethylene. The drinking of alcoholic beverages is believed to accentuate its action, while drinking milk possibly retards such action.

Procedures under Trichlorethylene Analgesia

Trichlorethylene analgesia is suitable for the following procedures: primary cleansing and débridement of burns; débridement of burn slough; open or closed venesection; manipulation of "frozen" joints; removal of imbedded or inaccessible sutures; painful dressings; shaving redundant burn granulations; reduction of simple fractures; removal of sequestra; incision and drainage of abscesses; suturing of lacerations; biopsies; dental extractions and other intraoral procedures; myringotomy; obstetrical deliveries; thoracoplasty; cystoscopy.

Advantages of Trichlorethylene

The advantages of trichlorethylene as an analgesic are several: The agent and apparatus are inexpensive. The inhaler is of small size and readily portable. The cylinder is nonspillable and essentially indestructible.

Potency of the vapor can be regulated, and analgesia is smooth and rapid without excitement during induction.

Trichlorethylene can be administered by the patient, whether child or adult, and momentary unconsciousness causes the mask to fall from the face when the agent is self-administered. Maximal safety is assured because the agent is nonflammatory and nonexplosive.

Premedication is not necessary. The use of trichlorethylene does not complicate or contraindicate the use of any other agent. There is a minimum of salivation or secretion of mucus, and nausea and vomiting are rare.

The agent can be used in the emergency room, the operating room, the patient's room or home, and the physician's office. If the patient is treated on an ambulatory basis, he can leave the hospital after twenty minutes. Metabolism of the liver and kidneys is not affected. There is no change in blood pressure, and capillary oozing is less noticeable than with other agents.

The agent does not depress respirations and has no effect on existing pulmonary lesions. The vapor also has no effect on the respiratory passages. As a general anesthetic it has been favored in the presence of pulmonary tuberculosis in the belief that respiratory tract irritation is minimal.

Disadvantages of Trichlorethylene

Trichlorethylene is not satisfactory for surgical anesthesia with muscular relaxation. It cannot be used in a closed system with soda lime, and it vaporizes with difficulty. Eye signs are unreliable. The use of trichlorethylene with epinephrine is dangerous. Tachypnea occurs if the rate of anesthesia is pushed too rapidly, and with rapid breathing overdosage is encountered. Bradycardia and arrhythmias may be induced.

Summary

Trichlorethylene has a definite place in the armamentarium of the anesthetist. We believe that it closely approximates an optimal agent for use in subjugating pain. If the anesthetist will respect the potency of trichlorethylene, he will find it a valuable aid in fulfilling his primary purpose: the relief of human suffering.

Controversial Questions in Anesthesia

A Forum

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Capt. O'Carroll: The purpose of this forum is to discuss controversial questions in anesthesia from a physiologic standpoint. There are differences of opinion even among the experts on many problems in the field of anesthesia. These differences involve certain technics, indications and methods for the use of drugs, and causes and proper treatment of complications. It is the purpose of this forum to explore some of these problems, with the object of arriving at a better understanding of the points in question and, where possible, to effect a compromise.

Subjects selected for discussion are: controlled respiration, balanced anesthesia, atropine, respiratory obstruction and laryngospasm, and muscle relaxants.

If any of you have any questions, we would like you to present your questions at the end of the program.

The first subject we are going to take up this morning is that of controlled respiration. Miss Miriam Shupp will be the first speaker. Miss Shupp.

Miss Shupp: During intrathoracic operations the major concerns are adequate ventilation and the least possible interference with circulation. Beliefs concerning the best technic of anesthesia for transthoracic operations are at variance. Therefore a number of technics are in current use, both with respect to anesthetic and method, but today we are primarily concerned with method.

Briefly, these technics are: (1) no change in method either before or after the pleura is opened, the patient being permitted to breathe at atmospheric pressure; (2) spontaneous breathing by the patient with pressure in the rebreathing

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circuit increased above atmospheric without any assistance to respiration; (3) maintenance of atmospheric pressure in the rebreathing circuit with respirations being assisted occasionally or continuously; (4) maintenance of pressures above atmospheric in the rebreathing circuit with respirations being assisted occasionally or continuously; (5) deliberate abolition of spontaneous breathing and complete control of breathing with respect to the rate, the volume, and the rhythm.

With the technic of controlled breathing the anesthetist may use either the hand or hand-knee to maintain respiratory movement (manual control) or use a mechanical respirator (mechanical control).

While it is my opinion, based on personal clinical experience and the work of other anesthetists, that a patient can safely be carried through a transthoracic procedure by allowing him to breathe spontaneously and by assisting respirations continuously with intermittent positive pressure, I prefer the technic of controlled breathing with the use of a mechanical respirator.

To the first question, "Is controlled breathing physiologically sound?" I would say it can be, and this I shall try to show.

When the pleural cavity is opened, if the pressure in the rebreathing circuit is kept at atmospheric and no measures are taken to aid the patient's respirations, so-called paradoxical breathing occurs, the mediastinum flaps back and forth with the patient's respiratory movements, and hypoxia and apnea result. The shifting and flapping of the mediastinum as well as the altered intrapleural and intrapulmonic pressure affect the mechanics of circulation, and the patient's condition rapidly deteriorates.

When the pleural cavity is opened, if continuous positive pressure is used and no assistance given to the respiration, the mediastinum may be stabilized, both lungs may be kept expanded, and oxygenation may be adequate (partially by diffusion respiration), but carbon dioxide is not adequately eliminated. Respiratory acidosis develops, and also cardiac output is reduced.

If continuous positive pressure is maintained within the rebreathing circuit and the respirations are assisted, tidal volume is increased, but there is an increased possibility of retained carbon dioxide and increased interference with circulation in reduced cardiac output.

The next technic to be considered is that of intermittent positive pressure assistance with the patient breathing spontaneously. This technic produces more normal variations in intrapulmonic pressures, which should result in more adequate oxygenation and more efficient elimination of carbon dioxide—and does, according to Beecher, Motley, Cournand and others—and also produces the least harmful effects upon circulation.

Watrous and others have gone further than saying assisted breathing should be used only in transthoracic procedures. According to Watrous, "controlled respiration or its modification, assisted respiration, . . . should be employed constantly in almost all inhalation anesthesias because it effectively counteracts the tendency of respiratory depressant and premedicant anesthetic agents to cause hypoxia or respiratory acidosis or both."

Motley, Cournand and others found that intermittent positive pressure interfered less with cardiac output than continuous positive pressure, and that certain types of respirators employing intermittent positive pressure interfered less than others, and that the difference was on the basis of the pressure curves. They found that the "ideal mask pressure curve should increase gradually during inspiration to a peak not higher than about 25 centimeters of water. The pressure should drop rapidly after reversal, in the beginning of expiration decreasing quickly to or near atmospheric with a mean mask pressure during the expiratory period as near as possible to atmospheric. Inspiratory time should not exceed expiratory time . . . this pressure curve permits the right side of the heart to compensate during expiration from the reduction in filling during inspiration."

If this is true, it would seem that if a patient were allowed to breathe spontaneously and the respirations were assisted by intermittent positive pressure, the mask pressure curve for optimal cardiac output could be altered and probably would be altered, depending upon the rate and rhythm of the patient's respirations. In assisted breathing the anesthetist can control volume only, not rate or rhythm.

Therefore, if the ideal mask pressure curve for maximal cardiac output is to be maintained, it would seem that control of rate, volume, and rhythm would be necessary. And the control of volume and rhythm as well as rate means the use of so-called controlled breathing.

If we use controlled breathing, shall we use mechanical or manual control? I have no idea what the average time for transthoracic operations might be, but in my experience they have lasted from several hours to ten hours. Can any anesthetist "squeeze" the bag, take the pulse and blood pressure and keep a chart, perhaps give drugs intravenously and manage fluid therapy for three or four or ten hours, and maintain the precision of the cycling curve, which has been shown to produce the most efficient cardiac output, efficient oxygenation, and as seemingly effective elimination of carbon dioxide as is possible with any other known technic?

In my opinion, the use of the mechanical respirator in transthoracic operations is a boon to the surgeon and the anesthetist as well as a benefit to the patient, but not many surgeons or anesthetists are aware of it yet.

As to the question, "Is controlled breathing essential to satisfactory operating conditions?" I feel that you would need to go to the surgeons and ask that question of them. But I am quite certain that the answer would be "Yes" from the surgeons who do use the controlled breathing technic and "No" from the surgeons who do not, and I think that those who answered "Yes" might also add that it depends upon the type of operation. I know that some of the delicate blood vessel and cardiac surgery that is done by Dr. Claude Beck of the University Hospitals of Cleveland would be extremely difficult without the quiet field produced with controlled breathing, either manual or mechanical.

"Does controlled respiration by manual or mechanical means alter the

carbon dioxide tension and content of the blood stream?" Beecher and associates reported a series of transthoracic cases in which the patient's respirations were unassisted except occasionally, and there was a greater incidence of respiratory acidosis in these cases than in the series of cases, reported by him and his associates a year later, in which respirations were assisted.

Gibbons reported a series of transthoracic cases with mechanically controlled breathing. His figures for arterial carbon dioxide tensions in the pneumonectomy group were comparable to Beecher's. The figures for arterial carbon dioxide tension were higher in the lobectomy group reported by Gibbons. Gibbons pointed out, however, that these figures might not be valid because of the small series and also stated that respiratory acidosis will develop in certain patients regardless of the technic of ventilation. Both men mentioned the need for more extensive research in this area.

"Does the complete apnea in controlled breathing (manual or mechanical) abolish the important prodromes of circulatory failure by disguising signs of overdosage?" The patient under controlled breathing, for some reason or reasons yet unknown — one, perhaps, being lessened metabolic rate because breathing is effortless on his part — requires much less anesthetic than the patient who is allowed to breathe spontaneously.

With the use of controlled breathing a new set of signs of anesthesia must be learned. The neophyte may very well overdose the first several patients when using controlled breathing technics. In my opinion, an anesthetist, even an experienced one, should not begin to use this technic without some practice under guidance or a sufficient number of observations of the work of others.

Our patients, unless there has been a cerebral accident, such as may occur in some types of cardiovascular operations, breathe within a minute or two after being taken off the respirator and are responding in the operating room. They are usually awake before they leave the operating room.

"Does mechanically controlled respiration increase the hazard of explosion? If so, how does the hazard compare with that in assisted or manually controlled respiration?" So far as I know, there are only two makes of respirators currently in use in the United States that can be used as part of the anesthesia equipment. One is the Mautz, which runs on air pressure, and from the standpoint of the explosion hazard — despite the fact that Beecher said mechanical respirators increase the explosion hazard — it presents no more of a hazard than the squeezing of the bag by hand.

The other respirator is the Rand-Wolfe, which is operated by an explosion-proof Fire Underwriters-approved motor. An electrical motor, even of the approved type, does increase the hazard, but no more so, I should think, than a motor-driven suction or ether-air machine.

"Does controlled respiration increase the incidence of pulmonary complications?" Not to my knowledge, although we have not done any studies on this. I believe the incidence is extremely low. Any technic that provides adequate ventilation, that prevents atelectatic areas from developing or remain-

ing, and that provides efficient circulation would, I should think, reduce the incidence.

Capt. O'Carroll: Dr. Hrant Stone will now give his point of view on the subject of controlled respiration. Dr. Stone.

Dr. Stone: The problem of controlled respiration has certainly changed in the past five or six years. Previously, there was a great deal of controversy whether or not controlled respiration was of value in the operating room under any circumstance. I believe today the feeling has gone to the other extreme where almost everyone feels that there are definite indications for using controlled respiration under certain circumstances.

Miss Shupp has certainly covered most of the important features of controlled respiration. I think, first of all, we should say something about the methods by which controlled respiration can be instituted.

During the course of an anesthesia the drugs that are used are certainly depressant to the respiratory center. The normal physiologic stimulus to respiration is carbon dioxide. Therefore, controlled respiration can be instituted, first, by increasing the resistance, or the threshold, of the respiratory center to carbon dioxide. This we do by the various anesthetic drugs we use, that is, it takes more carbon dioxide to stimulate the respiratory center than it did previously. Secondly, we may produce controlled respiration by washing out carbon dioxide, that is, by removing the physiologic stimulus, and hence the patient will become apneic, either until anoxia sets in or certainly until an active amount of carbon dioxide is allowed to accumulate. Third, controlled respiration of a different sort, a peripheral type of controlled respiration, can be instituted by the use of muscle relaxants.

During the course of clinical anesthesia one, two, or three of these technics are employed for producing controlled respiration. Probably, the most commonly used technic is to give a patient a depressing drug. We use morphine for medication, give cyclopropane, pentothal sodium, or other drugs that depress the respiratory center, and wash out carbon dioxide by compressing the rebreathing bag.

What are the possibilities of harm from controlled respiration? First of all, we must think of the rupture of an emphysematous bleb or the rupture of alveoli, which would allow air to escape outside of the lung perivascularly to produce mediastinal emphysema. Immediately, I should say it takes a great deal of pressure to cause such an accident, and usually during the course of clinical anesthesia such a pressure is not attained. The actual amount of pressure needed to rupture the lung is debatable, and no one has ever come up with a suitable explanation of the pressure needed to do it. It is possible, however, that in a patient with emphysema, an elderly patient, the use of positive pressure is attended by a greater hazard because of degenerative pulmonary changes, so that the lung has lost its distensibility, and rupture of the alveoli is a greater possibility.

Also, with a great deal of positive pressure that increases intrapulmonary pressure, you can drive oxygen into the arterial blood and produce arterial

embolism. Again, the amount of pressure needed to do such a thing is far greater than the pressure used during the course of clinical anesthesia.

So I believe you can immediately mark off the first two contraindications for, or dangers of, controlled respiration.

The third disadvantage to controlled respiration is the effect on the circulation. The first work that was done on the effect of positive pressure on circulation used constant positive pressure, that is, there was no phase during the course of the respiratory cycle when the intrapulmonary pressure returned to normal. At all times intrapulmonary pressure was above atmospheric pressure. If you use a technic of constant positive pressure, it is true that you can produce depression of circulation by positive pressure breathing. However, the technics in current use do not employ constant positive pressure. The technic that is used today is intermittent positive pressure with a relatively short phase of positive pressure; within a minute the amount of time given to positive pressure is much less than the amount of time during which relaxation takes place and atmospheric pressure exists in the lung. So if intermittent positive pressure is used with a long respiratory phase during which atmospheric pressure is reached within the intrapulmonary space, there is no significant effect on circulation.

Immediately I make an exception. If a patient with low blood pressure from shock or a decreased blood volume is put on positive pressure breathing, the circulation can be severely depressed. At the present time I do not know of anyone who has determined how much the systolic pressure must decrease before positive pressure breathing will produce detrimental effects. That certainly should be worked out. But let us say that a patient who is in fairly good condition will tolerate controlled respiration without any significant danger.

Miss Shupp covered the other features of controlled respiration, with the advantages and the disadvantages of using it. May I add that if a long intrapulmonary operation is undertaken without the use of controlled respiration, or at least semicontrolled respiration, the patient will have severe respiratory acidosis.

Miss Shupp pointed out that Gibbons and his group and also Beecher showed that while a patient can be adequately oxygenated—in fact, the oxygen saturation of hemoglobin increases during the course of an operation—carbon dioxide cannot be removed sufficiently for the prevention of respiratory acidosis unless some form of controlled respiration is used.

Capt. O'Carroll: From where I was sitting there didn't seem to be much controversy on that question. The next subject for the forum is balanced anesthesia, and I now introduce to you Mrs. Helen Lamb Powell.

Mrs. Powell: Just what is balanced anesthesia? I have not been able to find any satisfactory explanation of balanced anesthesia. Gwathmey, in the 1920's, combined the synergistic effects of morphine and magnesium sulfate with ether administered rectally for obstetrics. He called it synergistic analgesia. In other words, by the administration of drugs with synergistic actions, a less toxic dosage of any one drug was used.

Dr. Thomas, when I asked him what balanced anesthesia was, said, "Well, I always tell my people that balanced anesthesia is the anesthesia that the surgeon does not complain about."

Dr. Lundy was the first to call the administration of several drugs for premedication and anesthesia balanced anesthesia. His purpose was to gain an effect through a combination of agents that if produced by one agent would be undesirable.

We all know that ether is still regarded as one of the safest anesthetics. However, ether without the administration of premedication can be dangerous, with profound depression of respiration and circulation, anoxia, and other complications.

Nitrous oxide is also regarded as one of the safest anesthetics at our disposal, and no death has occurred from nitrous oxide if the patient received proper oxygenation. We all know the administration of nitrous oxide for the production of anesthesia without supplementing it with another anesthetic is very dangerous, but nitrous oxide in combination with another anesthetic drug is regarded as an ideal anesthetic in many clinics. The same may be said of ethylene.

Pentothal sodium like nitrous oxide is not a true anesthetic. It does not produce the relaxation necessary for intra-abdominal surgery. But in combination with one or more drugs it may be used to produce the degree of relaxation that will please the surgeon without profound depression. On the other hand, if we attempt to control the anesthesia with just pentothal sodium —without supplementing it with an inhalation anesthetic or some other drug—so much of the pentothal sodium must be given that the patient is profoundly depressed for days; there is likely to be deterioration.

Curare is one of the most valuable drugs that have been introduced into anesthesia in recent years. On the other hand, in some clinics today its use is forbidden, because of its misuse—its administration in profound doses. It has no direct effect upon circulation, but when a state of anoxia is brought about, that naturally depresses circulation.

With respect to current practices in regard to balanced anesthesia, I don't know that it could be called balanced anesthesia. You probably have heard the terms "garbage anesthesia" and "crash anesthesia." "Garbage anesthesia" and "crash anesthesia" are one and the same thing. It constitutes the administration of great dosages of premedication, then bringing the patient to the operating room and perhaps giving him a heavy dose of demerol, after which pentothal sodium is administered until he has not only lost consciousness but is in a profound state of respiratory and circulatory depression, with severe cyanosis before an inhalation anesthetic and/or curare is given.

So what is balanced anesthesia? It is the administration of a combination of drugs for anesthesia that will bring about as nearly a normal physiologic state during the administration of anesthesia as it is possible to achieve. Anesthesia is not a normal state. There are varying degrees of depression of both circulation and respiration and the danger of limitation of oxygen. So the

drugs we have at our disposal we should administer as carefully and as safely as possible. Profound depression of respiration should certainly not be countenanced by any anesthetist, nor should circulatory depression primarily due to the depressing action of the anesthetics. It behooves us all to have a thorough knowledge of the pharmacologic action of drugs used and avoid toxic drugs and dosages of drugs that will bring about depression.

CAPT. O'CARROLL: We will now hear from Dr. Krumperman, anesthesiologist at Temple University here in Philadelphia. Dr. Krumperman.

DR. KRUMPERMAN: It is very difficult to define balanced anesthesia. To me balanced anesthesia means a combination of anesthetic agents or anesthetic technics that will produce ideal operating conditions while the patient is maintained in as near a normal physiologic state as possible.

Balanced anesthesia has been abused, and abused badly, throughout the country, and I think when Beecher condemned "shotgun" anesthesia he had a perfect right to do so, because it has become a tendency everywhere to use dosages of drugs that are detrimental to the patient's well-being unless he is observed carefully. I do think that if this so-called shotgun anesthesia can be handled properly, there is no great damage done to the patient, but dosages and technics are all out of line in a fair number of places throughout the country.

When I think of balanced anesthesia, I usually do not think of the combination of various inhalation anesthetic agents but of the combination of general anesthesia with regional anesthesia. I also think balanced anesthesia should be reserved for the poor risk patient. For instance, a patient in the older age group is scheduled for a radical intra-abdominal operation for carcinoma. You feel that the best operating field would be produced by spinal anesthesia or epidural anesthesia, yet you know the patient's physiology would be greatly disturbed if the level were adequate to permit the operative procedure to be performed.

Therefore, if you could obtain abdominal relaxation and still take care of abnormal reflex disturbances and discomfort from traction that the patient would have if regional alone were used, this is the type of patient on whom to use a combination of regional and general anesthesia, whether it be with pentothal sodium, nitrous oxide, cyclopropane, ether, or ethylene.

A fair number of people have misused pentothal sodium with regional anesthesia as a form of balanced anesthesia. Pentothal sodium, as you know, is classified as an anesthetic agent, but its anesthetic properties are nil unless used in rather large doses. Therefore if a spinal or epidural block is poor and the surgeon happens to get out of the operative field and causes pain to the patient, the situation is rather difficult to control with pentothal sodium alone. If you have poor regional anesthesia, the proper technic to use to balance it would be some form of inhalation anesthesia. It could be pentothal sodium-nitrous oxide but not pentothal sodium alone. Or it could be any of the other general anesthetic agents that are available.

Curare is a wonderful adjunct to general anesthesia. But there again it is

an agent that has been misused and is now being abused by the surgical profession. Surgeons are blaming curare for a lot of the complications that are occurring not only in the immediate operative period but also in the post-operative period. We must use the muscle relaxants cautiously and not use them in too large doses. With the skill that most anesthetists should be able to attain after a few years of administering anesthesia, the dosages in which and the frequency with which these curare preparations are used can be minimized. They are needed, but I do think they should not be used to excess.

Surgical procedures for which I feel combined anesthesia is essential are those in the upper abdominal field, such as gastrectomies. For example, a surgeon may be unable to determine whether or not he is going to perform a subtotal or total gastrectomy until he has opened the abdominal cavity. If you have prepared for the major portion of the operation, which may be total gastrectomy, by administering spinal or another type of regional anesthesia and supplementing or complementing it with inhalation anesthesia, then there is no delay in the operation. The surgeon can progress from the abdomen into the chest, if necessary, without a lot of fuss, and it reduces the operative time for the surgeon and the patient, and therefore postoperative morbidity and possibly mortality will be lessened.

We should continue to use balanced anesthesia but be careful in the choice of the agents used and be careful to use it only on patients for whom we feel it is indicated. I don't feel that all patients need balanced anesthesia. I think it is a technic to be reserved for the poor risk patient.

Capt. O'Carroll: The next subject that we are going to take up is respiratory obstruction and laryngospasm. I will now introduce to you Miss Margaret Sullivan from the Roosevelt Hospital, New York City.

Miss Sullivan: Respiratory obstruction is the plague of all anesthesia, and I am certain that it is a condition that every person in this audience has not only encountered but has contributed to its production along with the accompanying complications.

Technic must be considered as a contributing factor in the production of respiratory obstruction. One of the questions submitted for discussion reads as follows: "Is any technic available that disguises the signs of respiratory obstruction?" To this question I must answer that some opponents of the mechanically controlled respiration technic cite it as being *the* one method in which the anesthetist is not aware of respiratory obstruction. From my own experience with mechanical control of respiration, which is by no means so extensive or varied as that of Miss Shupp, I think it is perhaps the *one* technic that presents the earliest signs or indications of respiratory obstruction. The anesthetist must very closely observe the operative field, or rather the inflation and deflation of the lungs. This technique offers the opportunity to note the lack of respiratory obstruction or, if such a condition should develop, to observe the first indication that all is not well within the lungs. Evidence of obstructing material, such as a tiny broncholith, is immediate with this technic.

I think that any anesthetic agent or adjuvant to anesthesia can be a contributing factor in disguising the signs of respiratory obstruction. Chloroform in

the lower planes of anesthesia, pentothal sodium, avertin, and curare and curare-like preparations all produce a diminished minute volume. This tends to invite respiratory obstruction.

The next question deals with the anesthetist. "In what way or ways does she or he contribute to the production of respiratory obstruction?" I think we could devote the greater part of this morning to a discussion of the role of the anesthetist in the production of this condition. The anesthetist who permits a high concentration of ether during induction, a concentration of cyclopropane that is too high, who fails to establish a patent airway with either a pharyngeal airway or an intratracheal catheter, who fails to remove secretions from the pharnyx, trachea, or bronchus, who fails to adjust the head and neck to support the jaw after the insertion of an airway is solely responsible for the obstruction that develops. These are but a few ways in which the anesthetist violates the principles of safe anesthesia.

Failure to institute prompt treatment is perhaps the greatest sin that can be committed in the presence of respiratory obstruction. We are well aware of the steps to be taken to right this condition. Some anesthetists resort to the use of drugs. It is my feeling that providing a patent airway, administering oxygen, and/or altering the level of anesthesia are the best methods to be employed. Atropine has been used for the treatment of laryngospasm that resulted in respiratory obstruction. Curare has no place in the treatment of respiratory obstruction, although this drug has been employed for the relief of laryngospasm.

To the question, "Is intratracheal intubation necessary to prevent respiratory obstruction?" I state with emphasis, "No."

The next question for consideration should be read in full: "How do agents, technics, the anesthetist, and the surgeon contribute to the production of laryngospasm? Are certain drugs, such as barbiturates, curare, atropine, and high concentrations of inhalation agents, or methods more prone than others to produce laryngospasm?"

Let us first consider the agents. It is an accepted fact that pentothal sodium does tend toward the production of laryngospasm, and it is likewise an accepted fact that laryngospasm usually occurs during the induction period. Why? Because this time finds the anesthetist involved in attending to so many details that his or her attention may be momentarily diverted, or the exact level of anesthesia may not be fully evaluated. Laryngospasm may occur during other periods of anesthesia, but only if the level of anesthesia is not sufficiently deep for the operation to be performed.

Atropine and curare are employed by some anesthetists for the relief of laryngospasm; thus we cannot consider them as drugs that aggravate the larynx.

It is my feeling that one of the greatest contributing factors in the production of laryngospasm is the pharyngeal airway. All too frequently the airway is inserted when the level of anesthesia is not sufficiently deep. Is there any anesthetist who has not been witness to the sequence of events that follows the insertion of a pharyngeal airway under light pentothal sodium anesthesia?

The surgeon contributes to the production of laryngospasm only insofar as

the anesthetist permits him to work under light anesthesia. I again refer to the agent pentothal sodium, for it is so frequently employed when laryngospasm does occur. We all have observed an attempted dilation of the rectal sphincter in the presence of light anesthesia, particularly when a barbiturate and some of the less potent agents have been employed. We all are aware of what follows. The same may be said for upper abdominal operations in the presence of light anesthesia. The agent, the technic, or the surgeon cannot be blamed; the anesthetist is entirely responsible. He or she is the person who should have full knowledge of the sequelae that will develop with each agent and technic.

The signs of oncoming laryngospasm may be overlooked. The level of anesthesia may not be correctly ascertained. Who or what is responsible? The anesthetist.

The anesthetist who fails to recognize the signs of obstruction or laryngospasm, who fails to appreciate the cause of either condition, who fails to institute immediate and adequate corrective treatment, be it the administration of oxygen or drugs, must assume full responsibility for all that ensues.

In conclusion, permit me to repeat the thought which I have been attempting to bring to you; not the agent, not the technic, not the surgeon, but the anesthetist is the one who either produces or prevents respiratory obstruction or laryngospasm.

Capt. O'Carroll: We are now going to hear from Dr. George R. Brighton, the attending endoscopist at the Roosevelt Hospital, New York City, and he will probably be able to tell us all about laryngospasm.

Dr. Brighton: I feel somewhat like a Taft supporter at an Eisenhower rally. I want of you and demand something of you. I am not only an endoscopist but also an otolaryngologist. That speaks for itself when you speak of laryngospasm.

Most of you may think of the larynx as the organ of phonation. Actually that is only one small part of its history. It goes back a long way in evolution. At the opening of the swim bladder of the fish are a group of muscles that act as a sphincter. That sphincter enables the fish to get away from its enemies because he can swim longer than his enemies can in water without oxygen. So remember that there is a psychologic reaction in laryngospasm. It is not all due to the anesthetist, as we have just heard. When you anesthetize a patient, you have a decerebrate animal, and that animal has a subconscious. In that subconscious is fear, and many times you will have a patient get laryngospasm in spite of the fact that you have been extremely careful in your induction and have used the most perfect technic and all of the drugs at your command. You still have laryngospasm. And I think to a large extent it goes back to the fear complex connected with the operation of the swim bladder of the fish. It may seem farfetched, but I do think there is something in that philosophy. We have never been able to prove the hookup with the nervous system, but it certainly does occur.

Laryngospasm is also due to mechanical obstruction. You get mechanical obstruction first, and then you get laryngospasm. We have made a revolutionary

change in the naming of some of our instruments. One of them is a tongue depressor. We now call that a tongue elevator, and you would be surprised what a difference it has made in laryngeal obstruction. I think you should go back to your operating rooms and simply tell your operating room supervisors that you no longer have tongue depressors; you have tongue elevators. It has made a tremendous difference in our technic both in anesthesia and in surgery.

Now, as Miss Sullivan asked, is there any technic of anesthesia that is a guarantee against respiratory obstruction? I say "Yes," there is a technic, and that technic is very careful induction. So many of us are in a hurry. We push our anesthetists and say "Come on, let's get this patient under. We don't have all day." This makes the anesthetist push the induction, which causes respiratory obstruction, which causes laryngospasm.

Miss Sullivan, I think, has touched on the way the anesthetist can help in removing the material that causes respiratory obstruction. The surgeon to a great extent can also help, because respiratory obstruction often occurs after the patient has been prepared by the anesthetist and the surgeon takes over, particularly the otolaryngologist. We are in the way most of the time, and we are apt to push the chin down or change the position of the head without paying much attention to the patient's respiratory efforts. It is difficult for the anesthetist to say to the surgeon, "You are causing obstruction."

As to the use of intubation to control laryngeal obstruction, I, for one, am completely opposed to routine intubation. I think anesthetists who say, "We use it on every patient we operate on," should be suspect.

The simpler technics in anesthesia can do away with laryngospasm and laryngeal obstruction only if they are properly applied and the proper anesthetic agents used.

There was one question, "Are drugs effective in the relief of respiratory obstruction or laryngospasm?" We have had some rather interesting experiences in doing laryngeal examinations under general anesthesia. The patient would be in third plane anesthesia, but the minute the laryngoscope was put into the larynx, the larynx would go into spasm. In cases of that sort topical anesthesia before general anesthesia will give you an anesthetized larynx with no reflex, so that you can have a fairly light general anesthesia and still be able to carry out the examination properly.

The problem of laryngospasm and laryngeal obstruction, particularly to us in otolaryngology and bronchoesophagology, is extremely important, and I appreciate very much the opportunity of talking to you this morning, because many times you and I have the same problem and have to work it out together.

Capt. O'Carroll: I am sure that all the people on the forum with me will agree with Dr. Brighton that the nervous system is hooked up with laryngospasm. I am sure we have all had some laryngeal irritation this morning even before we started to approach this microphone.

We will now hear something on the subject of atropine, and Miss Margherita Powers, from Johns Hopkins Hospital, will be the first speaker.

Miss Powers: "What are the effects of atropine on the respiratory func-

tions, circulation, the vagus nerve, and the heat-regulating mechanisms?"

Atropine has two separate actions affecting most of the organs of the body. The centers in the brain are stimulated, and the transmission of impulses from postganglionic craniosacral nerves to smooth muscle is interrupted. The specific effect on any organ or tissue is the function of the dose.

When administered as part of preanesthetic preparation, the dose of atropine is usually about 0.5 mg. In this dosage the respiratory center is stimulated, and the depressing effects of morphine or similar drugs are counteracted. In addition, the transmission of impulses from the vagus nerve to the bronchial muscles and the secretory glands of the respiratory tract is interrupted. Through these three effects, central stimulation, bronchial dilatation, and suppression of secretions, respiratory minute volume is increased.

Atropine has little direct action on the myocardium but causes cardiac slowing in doses of 1 mg. or less, by stimulating the nucleus of the vagus nerve. In persons in the extremes of age and in Negroes the cardiac slowing is less noticeable. With larger doses vagal inhibition is masked by the blocking action of atropine between the vagal terminations and the effector cells. Blood vessels, other than those in the splanchnic area, are dilated. The blood pressure is not usually altered by average doses.

Although peripheral vascular dilatation, under other circumstances, encourages heat loss, with atropine body temperature tends to increase because of the reduction in secretion of sweat and an increase in metabolism. Little evidence exists of direct effect on the temperature-regulating center.

"Is atropine effective against laryngospasm?" This has been covered fairly carefully in the previous discussion. The use of atropine to prevent and treat laryngospasm is widespread if one believes the literature. By reducing secretions atropine can be helpful in preventing spasms caused from local irritation of secretions. But as a treatment for laryngospasm there is no pharmacologic basis for its use. Atropine in the usually recommended doses has no effect on striated muscles. The muscles of the vocal cords are striated, even though they are innervated by the vagus nerve.

"In what types of cardiac disturbances is atropine indicated?" If atropine is to be used to treat cardiac disturbances, two points should be kept in mind: (1) Atropine prevents vagal inhibition of the heart whether reflex or direct. (2) Small doses (less than 1 mg.) will not interrupt completely impulses from the vagus. Effective treatment of sinus arrhythmias, ectopic beats, A-V blocks, premature systoles, and bradycardias, regardless of origin, is reported. However, some difference of opinion exists as to whether atropine is the best drug for any of these. Procaine, digitalis, quinidine, and epinephrine are also recommended. Internists and cardiologists have more faith in digitalis and quinidine usually.

"Does atropine as premedication disguise the signs of asphyxia?" Alterations in pulse rate, blood pressure, and respirations are generally considered the best signs of asphyxia. All of the drugs used as preanesthetic medication cause some variation in one or more of these. With atropine the onset of bradycardia may be delayed, and the blood pressure and respiratory changes may be abrupt

and slow in appearing. Little experimental work has been reported on this question. Reports of clinical observations are not very convincing, particularly when we remember that deep anesthesia also masks the same signs.

"Does the use of atropine contribute to the occurrence of convulsions?" By suppressing the secretion of sweat and increasing metabolism, atropine is possibly a factor in the production of convulsions. But it is only one of many factors. Inadequate exchange of respiratory gases, fever, high environmental temperature, malfunction of the temperature-regulating center, toxic disease, electrolyte and fluid imbalance, and the technic of anesthesia are all considered contributing factors. Some of these are beyond our control; others we can control to some degree. Before indicting atropine, we should, as far as we are able in every anesthesia, insure adequate exchange of oxygen and carbon dioxide, provide proper fluid replacement, and discourage heat retention.

"Does the use of atropine contribute to postoperative pulmonary complications?" We occasionally hear of a patient who has received too little or no atropine as premedication. In this patient the respiratory secretions were so profuse as to interfere with respiratory exchange and the establishment or maintenance of anesthesia. Whether the patient should be given atropine in such a situation is a controversial question. It has been stated that atropine will thicken the mucus, and that this will lead to atelectasis. It seems unreasonable that anyone would believe that atropine could have direct action on mucus bubbling in the lungs. However, as a result of inhibition of further secretion and bronchial dilatation, the increased airflow in the bronchial tree may lead to the evaporation of water from the mucus making it viscid. Deepening the anesthesia, if this can be accomplished, will produce the same effects. Aspiration of the pharynx or tracheobronchial tree is usually of very temporary or no help. If atropine is given, good postoperative care will probably prevent atelectasis.

"Should atropine be used routinely as premedication? If not, are there specific indications for its use?" The advisability of inhibiting the secretion of mucus and saliva during general anesthesia is probably unquestioned. It should be remembered, however, that scopolamine is a better drug for this purpose, although many consider it more toxic. Furthermore, the idea that the usual preanesthetic dose of atropine will reduce cardiac irregularities is unproved. On this basis, scopolamine may be substituted.

The use of either atropine or scopolamine before regional anesthesia causes the patient unnecessary discomfort in the form of an objectionably dry mouth. If general anesthesia becomes a necessity, atropine or scopolamine may be administered intravenously before beginning the induction.

Capt. O'Carroll: Our second speaker on the subject of atropine is Dr. Duncan A. Holaday, from the Presbyterian Hospital, New York City. Dr. Holaday.

Dr. Holaday: In 1940 Goodman and Gilman in their textbook outlined the experimental and clinical information on atropine. Its clinical use dates back to very ancient times when it was used for its mydriatic effects. Pharmacologic studies on atropine date back about a hundred years or more. And yet

there is a great deal that we don't know about it. Miss Powers has just reviewed for you much of what we do know about it, and I am going to take a minute to discuss some of the things that we don't know about it.

First, in brief recapitulation of what we do know. Atropine has two sites of action, central and peripheral. Centrally it stimulates most of the medullary centers and many higher centers. It may have some depressant action, as is indicated by its use in the treatment of parkinsonism, and yet this may not be a directly depressant action but a stimulating action having its effect by stimulating centers that inhibit the gross movements characteristic of parkinsonism.

Peripherally, it acts as if it paralyzed the postganglionic craniosacral nerves to smooth muscles and glands. It does not interfere with the production of acetylcholine. Apparently, as was indicated by the work of Clark in 1926, it competes with acetylcholine. Moe and collaborators in 1950 disproved the displacement theory of its action against acetylcholine in working with the pressor response to acetylcholine.

Atropine may have some direct effects upon myocardial tissue, although this is in dispute at the moment. It has some mild anticholinesterase activity, as was indicated by the work of Scholler in 1942. Marazzi in 1938 showed that it had some ganglionic blocking action. This has been confirmed by work that Koelle, Kamijo and I have done in recent months. So much for what we know about it.

We will consider for a moment what we don't know about it. In the first place, we have no idea as to what its mechanism of action is on the central nervous system. It has been the consensus until recently that synaptic transmission in the central nervous system was almost certainly not humoral or chemical and most probably electrical. If this theory obtains, there is little rational basis for believing that atropine has any effect centrally. However, di-isopropyl fluorophosphate and other anticholinesterases have been shown in recent years to have striking effects upon the central nervous system, and the evidence that a humoral mechanism may obtain is increasing. Atropine is useful in depressing the undesirable actions of di-isopropyl fluorophosphate, acetylcholine and eserine on the central nervous system. It may well be that it has a mechanism of action similar to that which obtains peripherally.

Regarding its peripheral mechanism of action, it would appear to act as curare does, that is to block the effects of liberated acetylcholine upon the receptor cells. That atropine primarily affects smooth muscles and glands, that curare blocks skeletal muscle, and that tetraethylammonium blocks ganglionic transmission is said to be due to the specificity of the receptor substance in the reacting end-organ.

That atropine efficiently blocks the action of acetylcholine at all muscarinic points of action but has a rather poor action upon the effects of vagal stimulation on the small intestine has been laid to the fact that the nerve endings going to the smooth muscle of the intestine are inside the cell at a site where atropine does not have access. This is mostly opinion, and there is little direct proof for it. Histologic studies of the smooth muscle of the intestine do little to clarify the point, the usual methods not being adequate to study these small structures.

However, certain histologic studies, including the work of Cajal, indicate that the nerve endings of the postganglionic nerves in the plexuses of the intestine do exhibit end plates that appear to be extracellular.

Another piece of evidence that may eventually help to clarify this matter has come from a French worker who has isolated buteryl choline from the smooth muscle of the bowel of the ox. It is perhaps that the normal transmitter of the postganglionic impulse in the intestine is not acetylcholine but some other choline ester, perhaps buteryl choline, and atropine may not be nearly so effective in competing with this transmitter as with acetylcholine.

Atropine is generally conceded to have no effect upon skeletal muscle. However, extremely large doses can be shown to modify the response to electrical stimulation of efferent motor nerves to skeletal muscle.

Regarding the problem of the efficiency of atropine in overcoming laryngospasm, I have no good opinion at all upon its clinical efficacy. I have tried it only once or twice. In one instance I thought it got me out of a rather difficult situation. I used two or three 1 mg. doses administered in rapid succession. I was using every other remedy at the same time, so I could not prove that atropine had any effect. Most people who say that atropine may have a beneficial effect upon laryngospasm have to rely upon the same kind of evidence. Nevertheless, the possibility has been raised, and there may be some basis in fact for it.

Two new facts have been uncovered recently that may eventually lead to an understanding of this question. Youmans at the University of Wisconsin described some very interesting experiments at the recent Pharmacology Society meeting. Stimulating the motor nerves directly in the severed sciatic, he was able to show that ether has a curareform action on a rather special preparation. He administered curare by slow drip to anesthetized dogs just to the point where there was slight interference with neuromuscular transmission. In such a preparation he could then administer ether and show a striking block of neuromuscular transmission to skeletal muscle at a time when the respiratory effects from the ether were just becoming apparent. This did not show up in every experiment. The one common denominator that he found in the study was atropine. In those 10 to 12 kg. dogs that had received premedication of 1/100 gr. atropine, he found an exquisite effect of this small dose of atropine upon the neuromuscular junction.

There may be effects, important effects, upon the larynx, which, although it is a striated muscle group, is not a muscle group having the same phylogenetic or ontogenetic derivations as most of the peripheral voluntary muscles, arising as it does from the branchiomeric muscle groups.

A second fact that may eventually help to clear up this particular problem concerns studies having to do with a rather obscure system going to skeletal muscles. Kuffler and his associates in recent years described the small fiber system going to skeletal muscles. Very briefly, in the frog this consists of nerve fibers half or less than half the diameter of the usual large alpha motor fibers that innervate skeletal muscles. Stimulation of these small efferent fibers in the frog's rectus produces a tonic response, not a twitch response such as one gets from stimulation of alpha motor fibers.

In mammals, the species we are primarily interested in, Kuffler was not able to demonstrate any motor responses to stimulation of these small nerves. They are present. He has described them in large representation in the sciatic nerve of the cat. The function that he ascribed to them is that they send motor impulses to one of the two primary proprioceptive organs in the muscles, namely, the muscle spindles. Impulses coming from muscle spindles which by monosynaptic pathways modify the response of the anterior horn cell to impulses playing on the anterior horn cell. An article by Tasaki and his associates raised the point that these may actually be motor fibers in the same sense that they are in the frog. Again I call your attention to the fact that the striated muscle of the larynx is not an ordinary group of musculature. Aside from its ontogenetic derivation, its function is considerably different from that of most of the skeletal muscles. It is an organ in which tonus plays a great role in its ordinary work. That normal tonus has anything to do with the tonus that we see in laryngospasm I don't necessarily mean to imply. However, the possibility certainly exists.

One more fact to bring out the point of this story. In the frog the motor fibers from anterior horn cells to the skeletal muscle of the rectus are exquisitely sensitive to blockade by curare. Atropine has very little effect upon neuromuscular transmission from these fibers. However, atropine does have a much greater effect in blocking the effects of stimulation of the small fiber system to this muscle.

I have not been able to find any information upon the effects of atropine on the small fiber system in mammals. I am not sure any work has been done.

This is a very provocative problem, and one that I hope more work will soon clear up. In the meantime there is good reason to keep open the question as to the efficacy of atropine on laryngospasm from a theoretical if not a clinical point of view.

Capt. O'Carroll: Our next subject will be muscle relaxants, and Mrs. Opal M. Schram, from Wesley Memorial Hospital, Chicago, will be the next speaker. Mrs. Schram.

Mrs. Schram: The problem of the use and misuse of the muscle relaxants never fails to bring forth innumerable varying opinions. The committee has offered eight questions concerning curare, and I shall introduce the discussion of this subject by presenting my answers to these questions.

The first question: "Under what conditions are muscle relaxants used as a substitute for adequate anesthesia?"

All of us are aware that muscle relaxants do not produce either narcosis or relief from pain, and hence that anesthesia is necessary. The difference of opinion apparently lies in the degree of anesthesia believed to be necessary. In our hospital we are accustomed to using upper second plane anesthesia for abdominal surgery. I am told that curare is used by some to keep the patient on the table with anesthesia so light that there is imminent danger of returning consciousness. This would constitute using the drug as a substitute for adequate anesthesia and would, in my opinion, be quite improper. On the other hand, to use curare to quiet a vigorous, muscular, young patient who is requiring an excessive amount of pentothal sodium seems to me justifiable.

The second question: "Does the respiratory depression following adequate curarization have ultimate ill effect?"

It is the responsibility of the anesthetist to maintain adequate respiratory exchange throughout anesthesia, and if this is done no harm will result during this period. It is the anesthetist's further responsibility to make as certain as possible that the respiratory exchange will be adequate during the immediate postanesthesia period, which means active, functioning intercostal muscles at the completion of the operation. Inadequate ventilation after operation should not be permitted. The literature reports postoperative atelectasis and death following curarization, but in most of the reports that have come to my attention curare was used in a more radical way than we are accustomed to using it. However, in some of these case reports the technic and dosage did not seem radical, so there may be occasional unusual delayed effects for which we should be on the alert. The article to which I refer is in *The Journal of the American Medical Association,* April 29, 1950, Foyger's article on "Fatalities following the use of curare."

"Are the adverse effects of muscle relaxants always reversible?"

One of the requirements of a good anesthetic drug is that its effects be reversible. Muscle relaxants qualify in this regard to about the same extent that anesthetic drugs do, with the exception that any drug given intravenously is attended by greater hazards because it is so easy to administer, and a gross error in dosage may be made. I would say that the adverse effects of muscle relaxants are reversible if the dosage is carefully individualized, and if the patient is not hypersensitive as is the patient with myasthenia gravis.

"Are the known antidotes to muscle relaxants always effective? Are the antidotes in themselves dangerous?"

The antidotes for muscle relaxants, as is true of antidotes for other drugs, may be ineffective and may be dangerous. While the careful anesthetist has little use for antidotes of any kind, the antidotes for muscle relaxants are comparatively safe and effective.

"What ill effects may result from repeated doses of muscle relaxants?"

d-Tubocurarine chloride, *d*-tubocurarine dimethyl ether, and flaxedil all have cumulative effects, and prolonged depression of the muscles of respiration may follow repeated doses. The greatest danger is present when the patient has left the care of the anesthetist and the reduced respiratory exchange may not be recognized by the attendant in charge.

Syncurine is not cumulative in action; on the contrary it becomes tachyphylactic, and repeated doses are ineffective in producing muscle relaxation. However syncurine has such pronounced depressing effects on respiration that this is a frequent complication following its use.

Judging from the reports in the literature, postoperative atelectasis is the complication we have to fear. In our hospital we use a muscle relaxant in approximately 1,800 out of 6,500 surgical cases per year and have been doing so for a number of years and so far have had no such complication.

"Are there dangers in the wide use of combined solutions of muscle relaxants and other agents?"

It is probably safe to assume that this question refers primarily to the combination of a muscle relaxant with pentothal sodium, such as the Baird solution or some modification of it. I cannot answer this question fairly as I am not accustomed to using a combined solution. If the patient's need for curare was always in direct proportion to his need for the sleep-producing drug, and if the two drugs were mixed in the proper proportion, the method should be effective. But if these variables were not all in balance, it would seem that there would be danger of inadequate or excessive effects of one or the other of the drugs. I know that those are fighting words, and I know many anesthetists use the combined solutions very successfully. As I say, I haven't had that experience.

"What are the dangers of histamine-like reactions of muscle relaxants? Which muscle relaxants are less prone to produce histamine-like actions?"

The histamine-like effects that are reported to attend the administration of muscle relaxants are decrease in blood pressure, bronchospasm, and bradycardia. In the experience of our anesthesia department such complications have been so rare as to be almost nonexistent. The antihistaminics, isuprel 1 cc. of 1:50,000 or aminophylline 250-300 mg., may be used to treat such reactions. Every new muscle relaxant that is introduced is said to have less histamine-like effects than its predecessor, hence syncurine, mytolon chloride, and succinyl choline are at present reported to have the least of such effects.

"What can be considered safe practice in the use of curare?"

The muscle relaxants should not be used by an anesthetist who is not sufficiently experienced to recognize inadequate respiratory exchange and compensate for it in any way that is necessary. Cautious, individualized dosage should be strictly adhered to, and the anesthetist should always be willing to change her plan if the reaction of the patient is unfavorable. The anesthetist should remember that curare is an adjunct to anesthesia, not a substitute for it.

If good judgment and common sense are employed in the use of curare, I believe that it can make anesthesia safer and more pleasant for the patient, more satisfactory for the surgeon, and easier and more satisfying to the anesthetist.

Capt. O'Carroll: I have a question here for Dr. Holaday: "Does atropine stimulate or depress the vagus?"

Dr. Holaday: Atropine depresses transmission of nerve impulses from the postganglionic craniosacral fibers of the vagus; it does nothing but depress at that site.

It has been said that small doses of atropine can stimulate the dorsal motor nucleus of the vagus. One may see bradycardia in some individuals. It is very difficult or impossible to demonstrate this in experimental animals. But in man it has been seen. The medullary-stimulating action on the dorsal motor nucleus of the vagus is overcome very easily by the peripheral blocking action. In any event it is usually very transitory, and this action disappears shortly after the dose has been given.

Dr. Stone: There is a question: "Do you use controlled respiration in thoracic surgery?" We do in almost every case that we do, yes.

"In your hospital, do you weigh sponges during operations? How is actual blood loss calculated by this method?" We weigh sponges in some instances. We do not do it routinely. Dry sponges are weighed, and the average weight of one dry sponge is calculated. Dry sponges are used during the operation, and each sponge is weighed after its use. The total weight of the wet sponge represents the sum of the weights of the dry sponge plus the blood it contains. The weight of the dry sponge, therefore, is subtracted from the total weight, which gives the weight of the blood. One gram of blood equals about 1 cc. Thus the volume of blood loss can be calculated.

Capt. O'Carroll: Question for Miss Shupp: "Is there not a real need at present for a positive-negative mechanical respirator that will respond to the needs of the patient when controlled breathing is indicated?"

Miss Shupp: There is a respirator, the E & J, that will deliver positive and negative pressure or positive pressure without the negative pressure. This respirator also will act as a demand or semi-demand type in cases where spontaneous respiration is resumed.

I may also state that research work is going on at the present time with negative-positive pressure anesthesia.

"If you were using an explosive gas along with a respirator with a motor—especially in pulmonary operations where cyclopropane is used—is using the machine advisable?" The respirator that uses air pressure should not be, and in our experience has not proved to be, any more an explosion hazard than manual pressure on the rebreathing bag. Any respirator that uses a motor, even though it is an approved motor, would, I should think, increase the explosion hazard. And if you had a leak in the rebreathing bag and had explosive mixtures of ether or cyclopropane or ethylene, you would increase the hazard. We are very, very careful that we use rebreathing bags that have no leaks in them.

Dr. Holaday: At Presbyterian Hospital we have a Rand-Wolfe respirator, which is motor driven. We use it all the time for thoracic operations with explosive mixtures, both cyclopropane and ether. We feel it is a very reliable machine as regards explosion hazard.

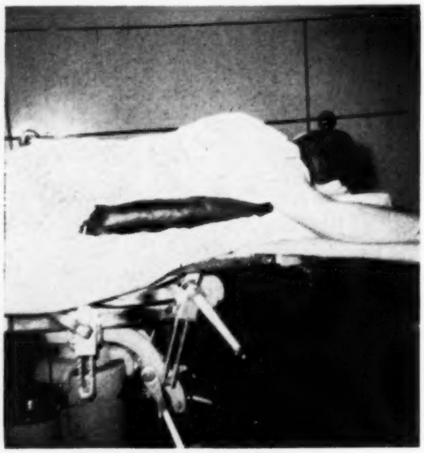
Dr. Stone: "Do you feel controlled respiration has a future place in upper abdominal operations without an open chest?" Yes. If you ever used controlled respiration for an upper abdominal operation and compared the conditions with those when a patient is breathing voluntarily, you would find a vast difference in the operating conditions. In one situation you have passive breathing, and in the other you have muscles that are actively contracting. There is also a lot more activity of the upper abdominal viscera. Controlled respiration gives a very quiet, relaxed operating field.

"What do you feel is the anesthetic of choice in most cases of thoracic surgery in adults and children?" I believe, and this is my own personal opinion, that cyclopropane is probably one of the best drugs to use. But of course you can use most any drug, and I should not say it is the best method. It certainly is a method which has a great deal of merit.

Notes and Case Reports

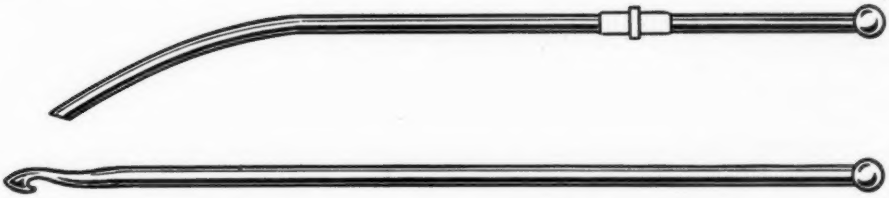
Comments on the Use of the Chandler Horseshoe for Operations in the Prone Position.—As all anesthetists have learned from experience, operations done in the prone position usually present the definite problem of maintaining a clear unembarrassed airway for the patient. Sandbags and many other devices have been used for years to support the patient and allow a clear respiratory exchange. The use of the Chandler "horseshoe" has almost entirely eliminated this problem for the author.

This "horseshoe" is a rubber-covered support that is filled with specially prepared horsehair, which does not tend to squash with weight upon it. It is distributed by V. Mueller & Co., Hospital Equipment Suppliers. It comes in three sizes, large, medium, and small, but I have found the medium size to be adequate for almost all patients. A very large patient may require some extra support from a small sandbag or two, but average patients, including children, fit the support perfectly. A face towel is used for a covering.



The patient is placed on the support so that the top of the "horseshoe" rests about one inch below the top of the sterum. The bottom parts of the "horseshoe" then support the sides of the chest cage, thus allowing a free respiratory exchange (see figures).—J. Pauline Benefiel, R.N., Indianapolis.

A Stylet for Intratracheal Tubes.—As every one knows in passing intratracheal catheters, it ofttimes becomes necessary to use a stylet. We have tried different kinds including copper stylets and pieces of welding rod. None of these was satisfactory because there was too much difference between the size of the stylet and the bore of the tube. In shopping around for something better, we came



Improvised stylet for intratracheal tubes.

across what seems to be the ideal stylet. It is made of aluminum and has a smooth surface. It is made from a "Boye" crochet hook, size J. We filed the hook off and bent it slightly. To keep it from slipping too far into the catheter a Telefon washer, which comes with each gas tank, was slipped on the right distance and taped in place. The stylet is adaptable to tubes size of a No. 7 Portex and larger. The accompanying illustration shows what it looks like originally and as a stylet.—**Hugh Stearns, R. N., and Forrest L. Pride, R. N.,** Hinsdale, Ill.

Sterilization of Drugs for Spinal Anesthesia.—Prompted by an article in *Anesthesiology* in regard to the sterilization of drugs for spinal anesthesia, the anesthesia department in this hospital adopted the method of autoclaving such drugs with the spinal sets at 240 F., 17 pounds pressure for twenty minutes.

In each set are placed those drugs that are most commonly used, namely, pontocaine crystals, 10 per cent dextrose solution, vasoxyl-procaine, and epinephrine ampules. In addition drugs that are used upon occasion, namely, heavy nupercaine, ephedrine sulfate, procaine crystals, pontocaine solution, are autoclaved in individual small jars and may be reautoclaved three or four times without affecting the drugs in any way.

This procedure has proved to be entirely satisfactory and saves an immeasurable amount of time. To quote Walton[1], the author of the article in *Anesthesiology*: "The above technique simplifies the procedure of spinal anesthesia, reduces the danger of bacterial contamination, and eliminates the decided danger of the antiseptic soaking solution entering a cracked ampule. If a neurologic complication does occur, at least one possible cause is automatically eliminated."—**Capt. Marian Waterhouse, A. N. C.,** Fort Devens, Mass.

1. Walton, F. A.: Correspondence to the editor. Anesthesiology 13:441, July 1952.

The eighteenth qualifying examination for membership in the American Association of Nurse Anesthetists will be conducted on Saturday, November 14, 1953.

The deadline for completed applications, including transcripts, is October 3. If application with transcript is received too close to the deadline, the application may not be processed in time for the candidate to be scheduled for this examination. The decision of the Credentials Committee will not be mailed to candidates before Oct. 5.

Legislation

Hospital Held Not Liable for Injuries to Patient During Electro-Shock Therapy[1] — An action was brought against a private hospital for damages for injuries suffered by a patient during electro-shock therapy. The physician administering the treatment was joined as a party defendant.

On appeal, the Appellate Division, Second Department, dismissed the complaint against the hospital because the evidence was insufficient to show that the hospital undertook to treat or did treat the patient; it merely furnished its facilities and a physician to administer the treatment pursuant to the directions of the patient's physician. The verdict against the physician who administered the treatment was against the weight of the evidence, and a new trial was granted as against him.

Scarring from Heat Treatment Was Not Necessarily Negligence[2] —A few days after she fell from a bicycle and bruised her right leg, an eight and one-half-year-old child was taken to a hospital. Her right leg was swollen and her temperature had risen to 104.8 degrees. Laboratory tests showed that she had osteomyelitis and that her blood stream had become infected with both staphylococcus and streptococcus germs.

Penicillin and "force fluids" were administered, compresses together with a heating pad were applied to the right leg. The heat treatments were discontinued when blisters began to form on the leg. The blisters developed into scar tissue.

At the trial, the jury chose to believe the evidence produced by the hospital that the scarring was a normal and natural result of the necessary treatment or of the osteomyelitis and blood poisoning, or that her condition was such that she was unable to stand the warmth necessary to save her life. The verdict was for the defendant-hospital.

Mental Suffering Caused by Baby Mixup Is No Basis for Damages[3]—On the third day after entering the hospital an obstetric patient had given to her an infant purported to be her own. She and her husband took the child home and cared for it until their discovery that it was not their baby. On making their grievance known the hospital delivered another child to the parents which they asserted was their baby.

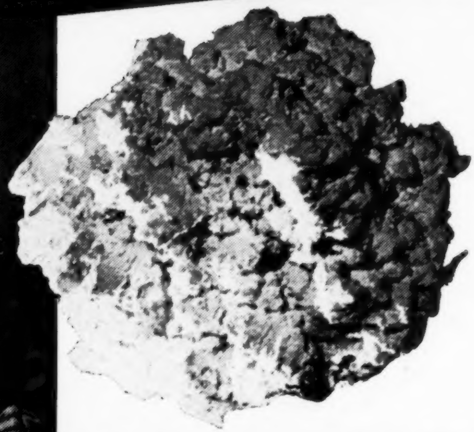
The parents sought to recover $50,000 damages resulting from negligence causing "profound shock to their nervous systems." A judgment in favor of the hospital was affirmed on appeal.

The court said that because there were no physical injuries to the parents, recovery for mental suffering could not be had. It is necessary that there be proof that the shock resulted in injury to the nervous system rather than to the mind.

Blood Donor Recovers for Injury After Having Blood Taken[4] —A professional blood donor who had given defendant a release or covenant not to sue, fainted and fell after

(Continued on page 201)

1. O'Rourke v. Halcyon Rest, App. Div. 2d Dept.; Mem., Feb. 9, 1953. (New York.)
2. Milias et al. v. Wheeler Hospital, 20 C.C.H. Neg. Cases 736, Calif.; March 17, 1952.
3. Espinosa v. Beverly Hospital, 1CCH Neg. Cases (2d) 598- Calif.; Nov. 17, 1952.
4. Boll v. Sharpe & Dohme, App. Div. 1; Ops.; 3-2, April 21, 1953. (New York.)



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Abstracts

ARCURI, R. A.; NEWMAN, W., AND BURSTEIN, C. L.: Electrocardiographic studies during endotracheal intubation. Effects during general anesthesia and hexylcaine hydrochloride topical spray. Anesthesiology **14**:46-52 (Jan.) 1953.

"In previous communications it was found that the incidence of electrocardiographic disturbances during endotracheal intubation under general anesthesia could be reduced from over 60 per cent to about 17 per cent with procaine given intravenously and to 12 per cent with diethylaminoethanol or procaine amide, respectively, administered intravenously prior to intubation. During the course of these investigations it was noted that topical cocainization of the pharynx and larynx seemed to enhance cardiac disorders, producing especially ventricular premature contractions. In an effort to overcome this peculiarity of cocaine to sensitize cardiac automatic tissue through stimulation of the sympathetic nervous system while retaining the beneficial features of local anesthesia, other topical agents have been tried. Unfortunately, some of these are also toxic in their own right, stimulating the central nervous system or depressing circulation. A new local anesthetic agent, 'cyclaine,' has been under investigation for some time and seems to be associated with a low index of toxicity as well as good topical anesthetic properties. . . .

"Electrocardiographic tracings were obtained in 50 adult patients who were given general anesthesia complemented by topical anesthetization of the larynx and pharynx with a 5 per cent aqueous solution of cyclaine. Electrocardiographic disturbances occurred in 14 per cent of the cases at the time of intubation. This incidence compares favorably with that in the previous series, including those in which procaine, diethylaminoethanol and procaine amide were used prior to endotracheal intubation. In the series in which procaine or its derivatives had been omitted under similar conditions the incidence of such disturbances was more than 60 per cent. Past experience has not helped to reduce the frequency or severity of these arrhythmias by using cocaine as a topical anesthetic agent. Cyclaine, a clinically new local anesthetic agent with adequate topical anesthetic properties, has been found to be free from sensitizing effects and, in addition, to possess a procaine-like action in actually clearing some arrhythmias produced."

BOYAN, C. P., AND BRUNSCHWIG, ALEXANDER: Hypotensive anesthesia in radical pelvic and abdominal surgery. Surgery **31**:829-838 (June) 1952.

"This report is concerned with the experiences gained in a series of 32 patients. . . . Hypotensive anesthesia produced by injection of hexamethonium bromide (C_6), a ganglionic blocking agent, and postural ischemia combined with pentothal sodium–*d*-tubocurarine–ether–oxygen anesthesia [was used]. . . . In order to fulfill one of the postulates for hypotensive anesthesia, good oxygenation of the blood, a patent airway was insured by endotracheal intubation and close observation of rate and depth of respiration. . . . Most of the procedures carried out were of more than usual magnitude and there was a certain quantity of

blood lost, which required immediate blood replacement in order to fulfill the main postulate of hypotensive anesthesia, namely, unreduced circulating blood volume. . . . There was no oliguria or anuria of significance in the postoperative course of the cases in this series. . . . The patients in this series were selected for unimpaired cardiovascular function. . . . Individuals not well sedated were noted to require larger amounts of C_6 to produce the desired hypotension. . . . It was probably a matter of coincidence that both 'failures' were Negroes. It is possible that the nervous stress in these individuals produced outpouring of adrenaline which counteracted the administered C_6. . . .

"When the blood pressure was returned nearly to normal before closing the abdomen, it was found that there were few bleeding points not previously noted during the hypotensive state. Apparently the time interval of hypotension was such that clotting occurred in those vessels. Dislodgement of clots by vigorous sponging of the operative field defeats this mechanism. The immediate postoperative period requires close supervision by experienced personnel to differentiate between internal hemorrhage with a drop in blood pressure and a prolonged response to C_6 administration. . . . It is the very distinct impression that these operations were appreciably facilitated and the blood loss reduced as a result of the hypotensive anesthesia."

Bunker, J. P.; Brewster, W. R.; Smith, R. M., and Beecher, H. K.: Metabolic effects of anesthesia in man. Acid-base balance in infants and children during anesthesia. J. Applied Physiol. **5**:233-241 (Nov.) 1952.

"In the normal adult man there is usually very little clinical evidence of a sympathetic response to ether anesthesia: tachycardia and tachypnea are not the rule; in fact their occurrence is cause for alarm. In infants and small children, however, elevated pulse and respiratory rates are regularly found during ether anesthesia. If this is a manifestation of increased activity of the sympathetic nervous system (or increased sensitivity to the products of such activity), then a metabolic acidosis might be expected during ether anesthesia in infants and small children even though it does not appear in adults. To test this hypothesis studies of acid-base equilibrium in infants and children were made during ether anesthesia. Parallel observations were also made during anesthesia with cyclopropane. . . . Studies were performed during ether anesthesia in 35 infants and children from 5 weeks to 12 years old. . . . Studies were carried out during cyclopropane anesthesia in 11 children aged 5 months to 6 years. . . .

"Serum pH, carbon dioxide content, and hemoglobin were determined in all cases, and in many cases serum lactic and keto acids, sodium, potassium, chloride, and inorganic phosphorus, and blood sugar were also determined. During ether anesthesia infants under 1 year, unlike adults, regularly developed a moderate metabolic acidosis (mean fall in corrected bicarbonate of 5.2 mEq./l.). A metabolic acidosis was observed in about 50 per cent of patients 1 to 12 years of age. This metabolic acidosis was regularly accompanied by a rise in serum lactate sufficient to account for more than half of the calculated change. There was no respiratory acidosis during ether anesthesia. During cyclopropane anesthesia in infants and children there was often, as in adults, a slight to moderate respiratory acidosis, but no metabolic acidosis. Serum potassium regularly fell during ether anesthesia and usually rose slightly during cyclopropane anesthesia. The changes in bicarbonate, lactic acid, blood sugar, and serum potassium during ether anesthesia are qualitatively and quan-

titatively similar to changes observed during the administration of epinephrine in physiological quantities. It is believed that these changes are produced by epinephrine released in response to a reflex stimulation of the sympathetic nervous system during ether anesthesia."

GABBARD, J. G.; ROOS, ALBERT; EASTWOOD, D. E., AND BURFORD, T. H.: The effect of ether anesthesia upon alveolar ventilation and acid-base balance in man; with particular reference to deficient ventilation and its prevention during intrathoracic procedures. Ann. Surg. **136**:680-690 (Oct.) 1952.

"It is the purpose of this work to report observations on the respiration of man anesthetized with one of the most common of anesthetic agents, ethyl ether. In an attempt to explain the changes in ventilation and acid-base balance observed with this agent, our data will be correlated with older ones obtained from human and animal experiments. The information thus gained will be applied to the prevention of some of the unfavorable side effects which have been ascribed to the drug. . . . Serious acidosis due to inadequate alveolar ventilation was always present in unassisted intrapleural operations. The acidosis could be completely prevented by assistance to ventilation. This ventilation had to exceed values considered 'adequate' for normal conscious man. No acidosis appeared in the extra-thoracic procedures; in fact, a moderate increase in alveolar ventilation and a respiratory alkalosis were often encountered. Ether per se in fourth plane, third stage of anesthesia had a stimulating effect upon ventilation and produced mild respiratory alkalosis. At this stage of anesthesia the respiratory center produced a normal ventilatory response to increasing carbon dioxide tensions up to 10 mm. Hg. above the resting value; beyond this value the sensitivity of the center to carbon dioxide decreased."

GREENE, B. A., AND BERKOWITZ, S.: The preanesthetic induced cough as a method of diagnosis of preoperative bronchitis. Ann. Int. Med. **37**:723-732 (Oct.) 1952.

"Three years ago we started asking each patient to cough during the first few minutes of spinal anesthesia to observe the ascent of muscular paralysis of the abdominal wall. We were surprised to hear a clearly 'wet' cough in many patients who had a normal respiratory tract according to the history and physical and roentgenologic examinations recorded in the hospital chart. When we extended the practice and requested a voluntary cough of every patient before the induction of any method of anesthesia, an apparent abnormality of the sound of the cough was quite commonly heard. It was not known, however, which of these sounds could be interpreted as evidence of tracheobronchial disease. . . . From September 1, 1950, to August 31, 1951, we examined 1,821 patients in an unselected and roughly consecutive series of elective and urgent cases in the operating rooms of two general hospitals serving largely the lower middle class income group. A majority was scheduled for elective surgery; this fact reduced the number with acute respiratory infections. All patients for tonsillectomy, pulmonary lobectomy and other operations performed because of respiratory disease were omitted from this study. We also studied several hundred other individuals; some were nonpatients and others were patients chosen to form special groups for separate consideration, as described later.

"Each subject was urged to cough vigorously unless an incarcerated hernia or ruptured viscus was suspected. The patient was asked to cough when he was lying on his side or sitting, because such a cough was more effective than if he was in the supine position. The cough was elicited early

RECENT STUDY PROVES VALUE OF "TRILENE" INHALATION ANALGESIA

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BROOKLYN, N. Y.—"Trilene" analgesia self administered with the "Duke" University Inhaler, offers an exceptionally high margin of safety for both mother and infant, and provides ease of administration, reported Charles E. Flowers, Jr., M. D., in a paper presented before the Brooklyn Gynecological Society.

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in the preanesthetic examination, before the patient had a chance to clear his respiratory passages consciously or unconsciously. The sound of the cough was evaluated at a normal conversational distance; the chest was not auscultated at the time. . . .

"An abnormality of the preanesthetic induced cough has been found to be the earliest significant sign of tracheobronchial disease. The patient with a normal induced cough just before anesthesia rarely develops a serious respiratory complication during the first three postoperative days, even after a gastric or biliary operation, in the absence of contamination of the lower respiratory tract with gastric contents. To avoid classifying an induced cough erroneously as normal, the observer must be experienced in the evaluation of the induced cough and in the consideration of the factors which interfere with the production of a vigorous cough effort. The patient with an abnormal induced cough, particularly if it is 'wet,' is very likely to have a postoperative cough or more serious respiratory complication, especially after an upper abdominal operation. The greatest value of the preanesthetic induced cough is in the diagnosis and selection of the patients who should receive special attention in order to prevent a major postoperative respiratory complication."

INGLIS, J. M.: Management of hypotension during anaesthesia. Lancet 2:363-367 (Aug. 23) 1952.

"Though in the modern practice of anaesthetics the combination of agents, the practice of administration, and the surgical procedures are numerous and often elaborate, there is a strong case for systematising the treatment of hypotension developing under anaesthesia. This is true especially where the onset is sudden; but whether the onset is sudden or not, it is generally agreed that such hypotension must be counteracted. Sometimes, in-

(Continued on page 206)

LEGISLATION

(Continued from page 195)

his blood had been extracted. He sued alleging want of care after his blood had been taken. The court held that the covenant not to sue was not broad enough to cover fall.

The omnibus clause which released the defendant against "all claims and demands whatsoever" under the ejusdem generis rule included only such matters particularly described in the instrument or those of a similar nature.

HOSPITAL NOT LIABLE FOR NEGLIGENCE OF ITS PROFESSIONAL STAFF, WITHOUT SUFFICIENT EVIDENCE[5]—An action was brought against the hospital for injury to a new-born infant. The child was born on August 10, 1950 and remained in the hospital until September 5, 1950 when he returned home with his mother. During his stay in the hospital, he was left with his mother for certain periods each day.

On September 12, 1950, the family physician discovered the infant had a fractured clavicle. Both the family physician and the X-ray specialist of the hospital agreed that the fracture was one week old on September 12, 1950. There was no question but that the fracture occurred in the hospital.

The patient was delivered by a doctor employed by the hospital which received payment for the delivery.

The court dismissed the complaint against the hospital as it was reluctant to presume administrative negligence which was necessary to make the hospital liable. Rather than presume the negligence in failing to exercise due care in the hiring of the medical staff and/or the non-medical help, the court ordered that the plaintiff must prove this administrative negligence before the hospital could be held liable.

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5. Ortega v. French Hospital, City Court, New York County, Kane, J., N. Y. Law Journal, March 24, 1953, p. 976.

Book Reviews

History of Anesthesia. By Virginia S. Thatcher. Cloth. 289 pages, 22 illustrations. Philadelphia: J. B. Lippincott Co., 1953. $5.00.

Virginia Thatcher, who is the editor of the American Association of Nurse Anesthetists publications, has put together in the *History of Anesthesia* that rare combination—a collection of historical facts plus a most readable, fast-moving account of the place of the nurse anesthetist in medical and hospital service. The development of the anesthetist as a nurse specializing in administration of anesthesia is traced through the development of anesthesia itself, placing it in its proper perspective, sociologically, economically as well as medically.

The dedication of the book "to the women who made anesthesia an art that it might become a science" is in effect the theme of the entire work. The first part outlines the coincident growth of medical and nursing education in the period following the discovery of anesthesia and the coming of age of both the well trained surgeon and the nurse. With ether as the anesthetic agent, its administration became an art, and the nurse a skilled professional in that art. As the possibilities of other drugs became apparent and anesthesiology took on a clinical entity, research went forward at a rapid rate. Anesthesia became a science, both pure and applied.

Miss Thatcher discusses the legal aspects of the nurse as anesthesiologist and nurse specialist. Through the first two parts of the book the story of anesthesia is interwoven with the story of changes in hospital development and practice. The administration of anesthesia is not discussed as a procedure in a vacuum but in relation to its use for patients in the hospital.

The last section of the book is devoted to organized nurse anesthesia—the beginning of the American Association of Nurse Anesthetists, its relationships with the American Hospital Association, the work of education, through schools, examination for registration and institutes.

No history is a true one unless it brings into focus the people who were most closely involved; Miss Thatcher has built much of her text around the individuals who have furthered the cause of anesthesia and those pioneers in this special field of nursing.

As a history and as a reference work, this volume is a welcome tribute to the field of anesthesia and one certain to promote new interest and enthusiasm.
—HOSPITALS, July 1953, Vol. 27.

The Pharmacology of Anesthetic Drugs. By John Adriani, M.D., Director, Department of Anesthesiology, Charity Hospital, New Orleans, Louisiana; Professor of Surgery, Tulane University School of Medicine; Associate Clinical Professor of Surgery, Louisiana State University School of Medicine. Ed. 3. Cloth. 179 pages, illustrated. Springfield, Ill.: Charles C Thomas, Publisher, 1952.

This current edition is an enlargement of the original edition published ten years ago. Recently acquired data have been added following laboratory or clinical investigation. The original form of the text has been retained; line drawings and charts being used liberally throughout. A glossary, tables, bibliographic references and an index of more than 7500 items adds to the

value of this book as a text for teaching and for a source of usable facts for the clinical anesthetist. The content is not restricted to anesthetic agents and pharmacologic facts but includes historical notes, actions of non-anesthetic drugs used in conjunction with anesthesia, clinical considerations of the actions of drugs as well as some chemistry and physics. This is a most valuable book in the field where constant investigation is bringing forth new information that will be useful to anesthetists.

The Anatomy of the Nervous System, Its Development and Function. By Stephen Walter Ranson, M.D., Ph.D., Late Professor of Neurology and Director of Neurological Institute, Northwestern University Medical School, Chicago. Revised by Sam Lillard Clark, M.D., Ph.D., Professor of Anatomy, The Vanderbilt University School of Medicine, Nashville, Tenn. Ed. 9. Cloth. 581 pages, 434 illustrations. Philadelphia: W. B. Saunders Co., 1953.

The original manner of presenting the anatomy of the nervous system with emphasis on the development and functional aspects has been continued in this ninth edition. New illustrations, new material and rewriting of portions of the text have been incorporated. Although written for the use of students of neuroanatomy, a great part of this book will be found useful to students of anesthesia.

Local Analgesia: Abdominal Surgery. By R. R. Macintosh, M.A., D.M., F.R.C.S. (Edin.), D.A., M.D. Nuffield Professor of Anaesthetics, University of Oxford, Civilian Consultant in Anaesthetics, Royal Air Force, Examiner for the D.A.; Anaesthetist, United Oxford Hospitals, and R. Bryce-Smith, M.A., B.M., B.Ch., D.A. First Assistant, Nuffield Department of Anaesthetics, University of Oxford; Anaesthetist, United Oxford Hospitals. At present on leave as Assistant Professor of Anaesthesia, Western Reserve University, Cleveland, Ohio. Cloth. 94 pages, 88 illustrations. Edinburgh and London: E. & S. Livingstone Ltd. 1953 (The Williams & Wilkins Co., Baltimore). $5.00.

Although the advent of curare into the field of anesthesia has made the indications for local analgesia less pressing than in the past, it is the authors' belief that the fact that local analgesics may be used only occasionally increases the need for clearly illustrated, simple instructions in the technics for their use. To this end they have prepared a small text based primarily on many illustrations. The technics in the book are limited to those suitable for use in abdominal surgery. Xylocaine, with its rapidity of onset of action, is the drug preferred by the authors.

The Use of Drugs. By Walter Modell, M.D., Assistant Professor, Cornell University Medical College, and Doris J. Place, R.N., Instructor in Medical Nursing, Cornell University, New York Hospital School of Nursing. Cloth. 468 pages, 8 illustrations. New York: Springer Publishing Company, Inc., 1953. $4.50.

This book is written for nurses and the material is selected and presented in a manner that will make the subjects easy to find and easy to understand. The four major divisions of the book indicate the wide range of subjects included: principles of pharmacology, principles of therapeutics, the medicine and materia medica. For the nurse who wishes to keep abreast of the changes that are taking place in the use of drugs in all branches of patient care, for the nurse who is interested in only certain phases of patient care and for the nurse who wishes to refresh her memory of dosages, arithmetic, weights and measures, this book would seem to have great value. For student nurse anesthetists, selected parts of this book should be invaluable in preparation for the study of drugs in relation to anesthesia. The chapters on the treatment of nervous system disorders among which are those relating to anesthesia, should be particularly useful to anesthetists.

Nutrition and Diet Therapy in Relation to Nursing. By Marie V. Krause, B.S., M.S., formerly Dietician in Charge of Nutrition Clinic and Associate Director of Education, Department of Nutrition, New York Hospital; Therapeutic Dietician and Instructor in Dietetics, Mount Sinai Hospital, Philadelphia, Pa. Cloth. 562 pages. Philadelphia: W. B. Saunders Co., 1952.

In this era of treating the "whole patient," it would seem that anesthetists, as well as nurses in other fields, will find this book useful in understanding the problems of nutrition. Of particular value would be the chapters on metabolic disturbances, the gastrointestinal system and the nutrition of surgical patients. Emphasis has been placed on applied nutrition rather than on tables and recipes as the basis for teaching this subject.

Simplified Arithmetic for Nurses. By M. Esther McClain, R.N., M.S., Instructor in Nursing Arts, Providence Hospital School of Nursing, Detroit, Michigan. Paper. 151 pages, illustrated. Philadelphia: W. B. Saunders Co., 1952.

A necessary part of the student nurse's training, the preparation of doses of drugs and of solution of drugs, is presented in this small book. For nurse anesthetists it may serve well as a review and reference handbook.

Side Effects of Drugs. By L. Meyler, Consulting Physician at Groningen, Netherlands. Translated by Ph. Vuijsje and W. Mulhall Corbet, Amsterdam. Cloth. 268 pages. Amsterdam: Elsevier Publishing Company, 1952 (Houston, Tex.).

The known facts regarding the side effects of drugs have been concisely presented with the author's hope "that it will be of use in investigating whether 'new' symptoms in the course of a certain disease are due to the disease itself or to the drugs used in its treatment." An extensive survey of the literature has been made to obtain the information pertinent to the subject of the book. Each chapter, in which specific groups of drugs are presented, is followed by references to the journals from which the information was obtained. The chapters on anesthetics, central nervous system depressants, central nervous system stimulants, blood derivatives and plasma substitutes are among the many that will be of interest to anesthetists.

ABSTRACTS

(Continued from page 201)

deed, this treatment is more important than continuation of the operation: for without it the patient's cardiovascular system will fail and the operation become pointless. . . . The development of hypotension during an operation under anaesthesia should be regarded as an emergency. Such hypotension can be divided into three groups: (1) with a raised pulse-rate; (2) with an unchanged pulse-rate; and (3) with a slowed pulse-rate. If the blood pressure falls suddenly, the patient's pulse-rate at that time indicates the immediate treatment. Hypotension with a raised pulse-rate is due to reduction of the cardiac output, largely because of reduction of the venous return. So the treatment aims at increasing the venous return by posture or by blood transfusion. Sometimes the cardiac output is reduced owing to insufficient contraction of the heart, perhaps due to overdosage with anaesthetic. Blood transfusion would then act too slowly, and treatment should be with cardiac analeptics and medullary stimulants. In both these forms of hypotension the treatment aims at increasing the cardiac output.

"Hypotension with unchanged pulse-rate is due to reduction of vasoconstrictor tone. Therefore treatment should be directed towards improving that tone with such drugs as methedrine and adrenaline. Hypotension with slowed pulse-rate is due to overstimulation of the vagus nerve, when the most effective treatment is pharmacological vagal block with atropine."

Nomination for Office American Association of Nurse Anesthetists

1953-54

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Edna Peterson (St. Francis Hospital, San Francisco); Graduate of St. Joseph's Hospital School of Nursing, Vancouver, Wash.; graduate of Sacred Heart Hospital School of Anesthesia, Spokane, Wash.; member of A.A.N.A. in good standing since 1941; former president, California Association of Nurse Anesthetists; former chairman, Western States Assembly of Nurse Anesthetists, member, Board of Trustees, A.A.N.A., 1950-52.

Harriet L. Aberg (Cottage Hospital, Galesburg, Ill.): Graduate of St. Luke's Hospital School of Nursing, St. Paul; graduate of Minneapolis General Hospital School of Anesthesia, Minneapolis; member of the A.A.N.A. in good standing since 1934; former vice president, New Jersey Association of Nurse Anesthetists; member, Publications Committee, A.A.N.A.; A.A.N.A. representative on Hospital Operating Rooms Committee, N.F.P.A.; member, Board of Trustees, A.A.N.A., 1950-52.

Anne Beddow (Crippled Children's Hospital, Birmingham, Ala.): Graduate of St. Vincent's Hospital School of Nursing, Birmingham; graduate of Lakeside School of Anesthesia, Cleveland; former president, Alabama Association of Nurse Anesthetists; member of A.A.N.A. in good standing since 1931; former president, Southeastern Assembly of Nurse Anesthetists; former president and executive secretary, Alabama State Nurses' Association; member, Board of Trustees, A.A.N.A., 1951-53.

TREASURER

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Marie A. Bader (Newark, N. J.): Graduate of All Souls Hospital School of Nursing, Morristown, N. J.; graduate of Mary Immaculate Hospital School of Anesthesia, Jamaica, L. I., N. Y.; member of A.A.N.A. in good standing since 1941; president New Jersey Association of Nurse Anesthetists.

Marie N. Bader (St. Mary's Hospital, Pueblo, Colorado): Graduate of St. Joseph's Hospital School of Nursing, Philadelphia; graduate of Postgraduate Hospital School of Anesthesia, University of Pennsylvania, Philadelphia; member of A.A.N.A. in good standing since 1939; former vice president, Colorado Association of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1946-48; 1st vice president, A.A.N.A., 1949-50.

Billie B. Caraway (Atlanta, Georgia): Graduate of Protestant Hospital School of Nursing, Fort Worth, Texas; graduate of Washington Park Hospital School of Anesthesia, Chicago; member A.A.N.A. in good standing since 1937; former president, Georgia Association of Nurse Anesthetists; former chairman, Southeastern Assembly of Nurse Anesthetists.

Jessie L. Compton (Dallas, Tex.): Graduate of Parkland Hospital School of Nursing, Dallas, Tex.; graduate of Baylor University Hospital School of Anesthesia, Dallas, Tex.; member of A.A.N.A. in good standing since 1940; former president, Texas Association of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1951-53.

Mary A. Costello (Cincinnati General Hospital, Cincinnati): Graduate of Mercy Hospital School of Nursing, Hamilton, Ohio; graduate of Cincinnati General Hospital School of Anesthesia, Cincinnati; member of A.A.N.A. in good standing since 1943; former president, Ohio Association of Nurse Anesthetists; member, A.A.N.A. Board of Trustees, 1949-51; chairman, Educational Fund Committee, A.A.N.A.

Nora W. Dell (Seattle, Wash.): Graduate of St. Joseph's Hospital School of Nursing, Tacoma, Wash.; graduate of Grace Hospital School of Anesthesia, Detroit, Mich.; member of A.A.N.A. in good standing since 1942; former president, Washington Association of Nurse Anesthetists; former chairman Western States Section of Nurse Anesthetists.

Josephine Handy (Roslindale, Mass.): Graduate of St. Elizabeth's Hospital School of Nursing, Brighton, Mass.; graduate of St. John's Hospital School of Anesthesia, Springfield, Ill.; member of A.A.N.A. in good standing since 1948; president Massachusetts Association of Nurse Anesthetists.

Martha S. Jackson (Municipal Hospital, Tampa, Fla.): Graduate of Macon Hospital School of Nursing, Macon, Ga.; graduate of Long Island College Hospital School of Anesthesia, Brooklyn; member of A.A.N.A. in good standing since 1943; former president, Florida Association of Nurse Anesthetists; former chairman, Southeastern Assembly of Nurse Anesthetists.

Alma D. Prykanoski (New Jersey State Hospital, Trenton, N. J.): Graduate of St. Francis Hospital School of Nursing, Trenton, N. J.; graduate of Mercy Hospital School of Anesthesia, Pittsburgh; member of A.A.N.A. in good standing since 1936; former president, New Jersey Association of Nurse Anesthetists; trustee, Middle Atlantic Assembly of Nurse Anesthetists.

Phyllis A. Roberts (Jefferson, Iowa): Graduate of Iowa Lutheran Hospital School of Nursing, Des Moines; graduate of University Hospitals School of Anesthesia, Cleveland; member of A.A.N.A. in good standing since 1941; former president, Iowa Association of Nurse Anesthetists; secretary, Upper Midwest Conference of Nurse Anesthetists.

Kathryn C. Svoboda (Omaha, Nebraska): Graduate of St. Catherine's Hospital School of Nursing, Omaha; St. Catherine's Hospital School of Anesthesia, Omaha; member of A.A.N.A. in good standing since 1936; former secretary-treasurer, Nebraska Association of Nurse Anesthetists.

Twentieth Annual Convention American Association of Nurse Anesthetists

August 31 — September 3, 1953
CIVIC AUDITORIUM
SAN FRANCISCO

PROGRAM

Sunday, August 30

10:00 A.M.—6:00 P.M.—**Registration**
A.H.A. Headquarters — Palace Hotel

9:00 A.M.—**Assembly of Directors of Schools of Anesthesia**
French Room — Sir Francis Drake Hotel
Helen Vos, R.N.
Director, Hurley Hospital School of Anesthesia
Flint, Mich.
Class Outlines for Schools of Anesthesia

2:00 P.M.—**Assembly of Directors of Schools of Anesthesia**
Helen Lamb Powell, R.N.
Chairman, Education Committee
Presiding Officer

Student-Teacher Relationships in Schools of Anesthesia
Cameron W. Meredith, Ph.D.
Professor of Education
Northwestern University, Evanston, Ill.

Monday, August 31

8:00 A.M.—**Registration**
A.H.A. Registration — Registration Desk, Civic Auditorium
A.A.N.A. Registration — A.A.N.A. Service Booth 2000

9:00 A.M.—**Assembly of Directors of Schools of Anesthesia**
Grove Room, Civic Auditorium
Lucille M. Lovett, R.N.
A.A.N.A. Educational Director
Teaching Technics

1:45 P.M.— **General Session**

Grove Room, Civic Auditorium
Josephine B. Bunch, R.N.
President, A.A.N.A.
Presiding Officer

Invocation
Sister M. Catherine
Mercy Hospital
Redding, Calif.

Address of Welcome
Josephine B. Bunch, R.N.
President, A.A.N.A.

Address of Welcome from A.H.A.
George Bugbee
Executive Director
American Hospital Association

2:30 P.M.— Mrs. Ellenor M. Penrose, R.N.
Salt Lake City, Utah
Presiding Officer

Pediatric Anesthesia
Lloyd H. Mousel, M.D.
Director of Anesthesia
Swedish Hospital, Seattle, Wash.

Surgical Problems of the Crippled Child — Paper with Motion Picture
(Josephine Bunch, Participating)
Leo S. Lucas, M.D.
Portland, Ore.

Psychological Preparation of Children for Tonsillectomy (with Special Reference to Anesthesia)
Francis A. Sooy, M.D.
Associate Clinical Professor, Otolaryngology
University of California School of Medicine
San Francisco, Calif.

Anesthesia for the Crippled Child
Anne E. Beddow, R.N., Anesthetist
Crippled Children's Hospital
Birmingham, Ala.

7:00 P.M.—**State Night Dinner**
Ballroom, Palace Hotel
Edna Peterson, R.N.
Chairman, Convention Committee
Presiding Officer

Tuesday, September 1

9:00 A.M.— **Business Session**

Grove Room, Civic Auditorium
Josephine Bunch, R.N.
President, A.A.N.A.
Presiding Officer

Call to Order
Report of Approval of Minutes Committee
Reports of Officers
Reports of Standing Committees

11:00 A.M.— **Election of Officers**

2:00 P.M.— **Business Session**

Grove Room, Civic Auditorium
Josephine Bunch, R.N.
President, A.A.N.A.
Presiding Officer

Reports of Standing Committees
Reports of Special Committees
Unfinished Business
New Business

Wednesday, September 2

Clinics at San Francisco-Oakland-Berkeley Hospitals

St. Francis Memorial Hospital
St. Mary's Hospital
Letterman Army Hospital
Hahnemann Hospital
Providence Hospital
San Francisco Hospital
Southern Pacific Hospital
Veterans Administration Hospital
Samuel Merritt Hospital
Alameda County Hospital
Children's Hospital of East Bay
Kaiser Foundation Hospital
Herrick Memorial Hospital

2:00 P.M.— **General Session**

Grove Room, Civic Auditorium
Ursula Heitmeyer, R.N.
President, California Association of Nurse Anesthetists
Presiding Officer

Recent Developments in Cardiac Physiology in Relation to Anesthesia
Lt. Col. Arthur B. Tarrow, USAF, M.C.
Director of School of Anesthesiology
Lackland AFB, Texas

Pulmonary Physiology: Its Importance in Anesthesia for Thoracic Surgery
Major Harry P. Makel, M.C.
Chief Anesthesia Section
Letterman Army Hospital
San Francisco, Calif.

Recent Developments in Endocrine Physiology in Relation to Anesthesia
John E. Cann, M.D.
Anesthesiologist, San Francisco, Calif.

Pharmaco-Physiologic Effects of Muscle Relaxants and Their Antagonists
Robert W. Virtue, M.D.
Associate Professor of Anesthesiology
University of Colorado Medical Center
Denver, Colo.

7:30 P.M.— **Banquet**

Gold Room, Fairmont Hotel
Josephine B. Bunch, R.N.
President, A.A.N.A.
Presiding Officer

Invocation
Rev. Father Chas. R. Hackel
Executive Director
Catholic Social Services
Oakland, Calif.

Where Do We Go from Here?
Robert R. Gros
Pacific Gas & Electric Company
San Francisco, Calif.

Presentation of A.A.N.A. Award of Appreciation
Agnes M. McGee, Receiving
St. Vincent's Hospital
Portland, Ore.

Thursday, September 3

9:00 A.M.— **General Session**

Velta Rogers
President, Utah Association of Nurse Anesthetists
Presiding Officer

Hypoventilation

Carl W. Fisher, M.D.
Chief Anesthesiologist
Kaiser Foundation Hospitals
Oakland, Calif.

Clinical Anesthesia vs. Textbook Anesthesia

John Adriani, M.D.
Director, Department of Anesthesia
Charity Hospital
New Orleans, La.

2:00 P.M.— **Council Session**

Florence A. McQuillen, R.N.
Executive Director, A.A.N.A.
Presiding Officer

Visiting of Exhibits

Unfinished Business

Adjournment

Classified Advertisements

NURSE ANESTHETIST: For approved pediatric hospital. Write: Administrator, Milwaukee Children's Hospital, 721 North 17 St., Milwaukee 3, Wis.

NURSE ANESTHETIST: 650 bed fully equipped general hospital. Salary range: $363-$449; forty hour week; one month paid vacation. Write: Personnel Director, Jackson Memorial Hospital, Miami, Fla.

NURSE ANESTHETISTS for a 150 bed hospital. Forty-five hour week; extra pay for call duty; salary $425 a month; vacation; holidays; sick leaves. St. Joseph's Hospital, Minot, N. Dak.

WANTED: Nurse anesthetist; 75 bed modern general hospital, fully approved, located in a city of 12,000 population. Salary $400 per month and complete maintenance. Apply to: Administrator, Radford Community Hospital, Radford, Va.

NURSE ANESTHETIST: Wanted immediately for established general hospital of 100 beds. Liberal employee benefits. Write for information and personal interview to: Sister Charles Auguste, Providence Hospital, Anchorage. Alaska.

WANTED: Nurse anesthetist; fully approved 70 bed hospital with well qualified clinic group as active staff. Salary above average for Middle Atlantic area. Write: Box M-16, Journal A.A.N.A., 116 S. Michigan Ave., Chicago 3, Ill.

NURSE ANESTHETISTS for 126 bed hospital. For particulars contact: Director of Nurses, Eye, Ear, Nose & Throat Hospital, 145 Elk Place, New Orleans, La.

WANTED: Nurse anesthetist for 58 bed hospital; mostly surgical patients; excellent staff of surgeons. Good hours; salary open. Please contact: Irene Weaver, R.N., Administrator, The Gaston Hospital, 3505 Gaston, Dallas, Tex., TEnison-8101.

ANESTHETISTS: Three; 100 bed acute general hospital; large volume of surgery. Pleasant working environment, modern equipment, air conditioned operating room, forty hour week except for every third week, vacations, sick leave, Social Security; small town in East. Salary $350 per month. Apply: Box M-18, Journal A.A.N.A., 116 S. Michigan Ave., Chicago 3, Ill.

POSITIONS AVAILABLE—ACTIVE TEACHING HOSPITAL: Salary range from $300 to $400 plus, depending on experience and ability. Five day week; no obstetrics; little night work. G. Bittenbender, Associate Professor of Anesthesiology, Baylor University College of Medicine, Houston, Tex.

WANTED: Surgical anesthetist for 150 bed general hospital; central Nebraska. Excellent working conditions and personnel policies. $450 per month and full maintenance. Apply: Box E-70, Journal A.A.N.A., 116 S. Michigan Ave., Chicago 3, Ill.

NURSE ANESTHETIST: 350 bed approved general hospital twenty minutes from New York City. Salary from $260 to $320 per month with full maintenance. Forty hour week; special bonus for call duty; average one night per week call. Four weeks' vacation plus seven paid holidays. Department headed by anesthesiologist. Write: Box M-12, Journal A.A.N.A., 116 S. Michigan Ave., Chicago 3, Ill.

WANTED: Nurse anesthetist. 300 bed hospital, attractive employment conditions, salary open. Reply stating age and experience to J. M. Schwab, M.D., Chief of the Department of Anesthesiology, Geisinger Memorial Hospital and Foss Clinic, Danville, Pa.

ANESTHETISTS (3): Immediate openings available. Hospital located in New York City. All types of surgery (no obstetrics). Salary $4800 to $6000 per year. One month vacation at end of each year; two weeks' sick leave per year. Quarters available in residence ($425 annually). Apply: Box M-10, Journal A.A.N.A. 116 S. Michigan Ave., Chicago 3, Ill.

WANTED: Nurse anesthetists for 500 bed University teaching hospital; starting salary $383 per month. Stated increases; vacation and holiday leave; cumulative sick leave. Apply: Anesthesiologist in Charge, University of Virginia Hospital, Charlottesville, Va.

TWO NURSE ANESTHETISTS to increase staff, allowing better hours. Good salary; pleasant working conditions; complete maintenance if desired. Approved general hospital west side Chicago. Apply: Box M-20, Journal A.A.N.A., 116 S. Michigan Ave., Chicago 3, Ill.

MOVING TO PHILADELPHIA? Suburban general hospital, 130 beds, needs a nurse anesthetist in addition to present staff. No night OB calls. Write: Administrator, Chestnut Hill Hospital, 8835 Germantown Ave., Philadelphia 18, Pa.

ANESTHETISTS WANTED: Busy suburban hospital near Chicago. New nurses' residence (apartments available for married anesthetists). Starting salary $315 per month and complete maintenance which is equivalent to $450. After sixty days $330 per month. Vacation, sick leave, Blue Cross insurance and free life insurance. MacNeal Memorial Hospital, Berwyn, Ill.

ANESTHETIST wanted at once to increase personnel in well organized department of anesthesiology in a 300 bed hospital located in Wilmington, Delaware. Salary $350 a month, with meals and laundry. For details write: Dr. Richard E. Allen, The Memorial Hospital, Wilmington, Del.

WANTED: Nurse anesthetist. 100 bed approved general hospital; good working conditions with maintenance if desired. Salary open. City of 10,000 population. Apply to: Administrator, Davis Memorial Hospital, Elkins, W. Va.

MOVING TO MILWAUKEE? Position for nurse anesthetist in 350 bed teaching hospital with residents and interns. Present staff of eight nurse anesthetists and three medical anesthetists. Work averages 32 hours per week plus 24 hours additional on call. Salary open; month's vacation with pay; other benefits. Apply to: Administrator, Milwaukee Hospital, 2200 W. Kilbourn Ave., Milwaukee 3, Wis.

WANTED IMMEDIATELY: Nurse anesthetist. Department covered by twelve with 7,000 anesthesias per year. Apply: Director of Anesthesia, Henry Ford Hospital, Detroit, Mich.

NURSE ANESTHETISTS (A.A.N.A. members): Starting salary $365; automatic increases; laundry of uniforms. Forty hour week. No obstetrics. Liberal vacation and personnel policy. SUTTER HOSPITAL OF SACRAMENTO, CALIF.

Wanted Immediately!

NURSE ANESTHETIST

is wanted immediately for a completely modern, 109-bed general hospital in the Pacific Northwest.

The hospital, A.C.S. approved, is operated by General Electric Company in Richland, Washington. Two full-time nurse anesthetists already are on duty.

Salary is good, and liberal employee benefits include paid vacations and holidays, low-cost health and life insurance, pension plan, suggestion award system, plus many others.

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Apply by collect wire or air mail letter to

ADMINISTRATOR,
KADLEC HOSPITAL
Richland, Washington

NURSE ANESTHETIST: Apply: Director of Anesthesiology, Abington Memorial Hospital, Abington, Pa.

WANTED: Well qualified nurse anesthetist to work with clinic group. Salary open, based on experience; all types of surgery; two hospitals associated with group, with total capacity of 150 beds. Carbondale Clinic, Carbondale, Ill.

WANTED: Two nurse anesthetists for 240 bed hospital. Salary open. Partial maintenance provided. Apply: Administrator, Charleston General Hospital, Charleston, W. Va.

IMMEDIATE OPENINGS AVAILABLE: A.A.N.A. members, two nurse anesthetists needed. Obstetric anesthesia in a very active department with 350 to 400 deliveries monthly. Eight hour rotating shifts. $350 a month beginning salary with room and laundry; 50 per cent of anesthesia fee per case for second call. Social Security, and very pleasant working conditions. Apply: Administrator, Good Samaritan Hospital, Dayton, Ohio.

ONE NURSE ANESTHETIST: For 125 bed general hospital. Salary open. Full maintenance. Apply to: Superintendent, Maine Eye and Ear Infirmary, Portland, Maine.

WANTED: Nurse anesthetists for 800 bed teaching hospital. Staff of three anesthesiologists, three residents, ten nurse anesthetists. Cash salary $4606.08 annually with merit rating increases. One month's paid vacation, fifteen days' sick leave annually which can accumulate to ninety days. Only emergency operations on Saturday. Please reply to: Anesthesia Department, Medical College of Virginia, Richmond 19, Va.

NURSE ANESTHETIST: Approved hospital near Detroit. $450 per month. Overtime after forty hours per week. Living quarters available. Wyandotte General Hospital, Wyandotte, Mich.

NURSE ANESTHETIST for 236 bed general hospital 30 miles from New York City. Live out. Salary open. State age, training and experience. Morristown Memorial Hospital, Morristown, N.J.

NURSE ANESTHETIST: With adequate experience to assume some supervisory duties in addition to administering anesthesia. Salary open. Apply: Chief, Anesthesia Department, The Mercer Hospital, Trenton, N. J.

NURSE ANESTHETIST to supervise postoperative recovery room in new 240 bed hospital to be opened October, 1953. Capable of setting up this service. Days only; no call; salary open. Write: Personnel Director, The Methodist Hospital, 119 W. Lewis Street, Fort Wayne, Ind.

WANTED: Nurse anesthetist for 100 bed cancer hospital, active major surgical service, congenial working conditions, excellent equipment, very little call, forty hour week, salary open. Apply: Medical Director, Ellis Fischel State Cancer Hospital, Columbia, Mo.

NURSE ANESTHETIST for approved 113 bed hospital. Good salary, vacation, sick leave and Social Security. Excellent location near Chicago. Apply: Personnel Director, Highland Park Hospital, Highland Park, Ill.

NURSE ANESTHETIST: To increase present staff of 10 nurse anesthetists. A.A.N.A. membership or eligibility for membership required. Starting salary $375 per month with yearly increase, plus full maintenance or cash allowance for maintenance, as desired. Private room with bath and telephone in new women's residence. Social Security and private pension plan. Excellent working conditions; surgical and delivery suites air conditioned. 44 hour week. Apply: Marshall Kerry, M.D., Chief Anesthesia, The Reading Hospital, Reading, Pa.

ANESTHETIST: M.D. or R.N.; male or female; registered; experienced. No night call; uniforms and laundry; $450 beginning salary. 200 bed general hospital. For information write: Administrator, Leila Y. Post Montgomery Hospital, Battle Creek, Mich.

NOTICE

To all graduates of Grace

San Francisco Meeting

Annual Breakfast

Tuesday, Sept. 1, 1953

Time: 7:30 A.M.

Place: Hotel California

Reservations by Monday, August 31st:

Contact Dorothy Case *or* Mabel E. Courtney

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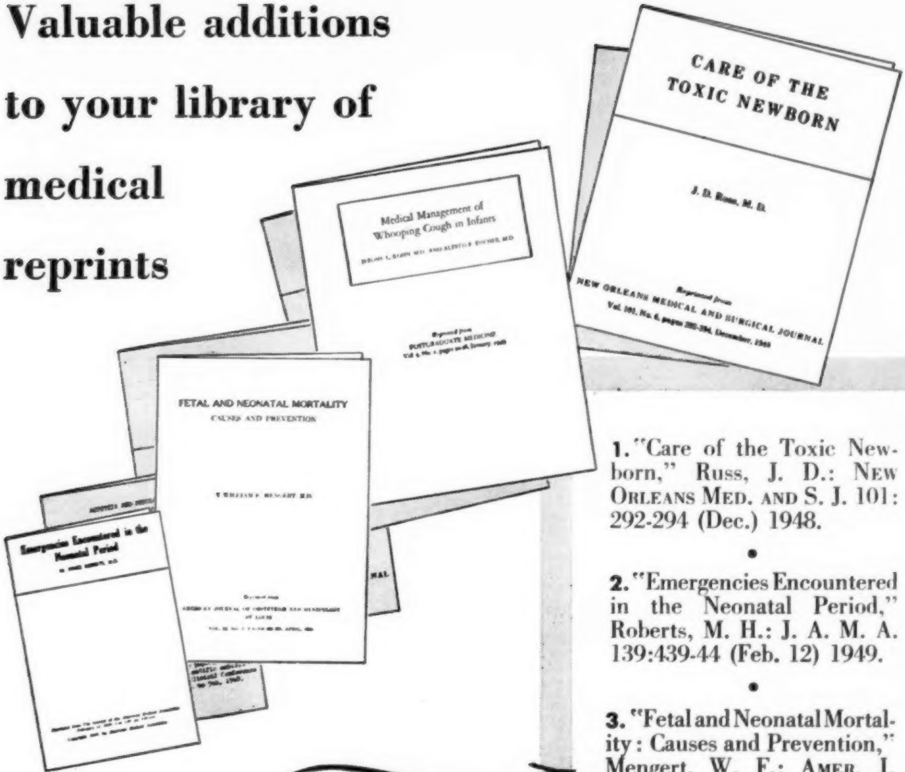

These articles, which discuss the use of oxygen in medicine, are available from LINDE's oxygen therapy department.

1. "Care of the Toxic Newborn," Russ, J. D.: NEW ORLEANS MED. AND S. J. 101: 292-294 (Dec.) 1948.

•

2. "Emergencies Encountered in the Neonatal Period," Roberts, M. H.: J. A. M. A. 139:439-44 (Feb. 12) 1949.

•

3. "Fetal and Neonatal Mortality: Causes and Prevention," Mengert, W. F.: AMER. J. OBST. AND GYN. 55:660-668 (April) 1948.

•

4. "Medical Management of Whooping Cough in Infants," Kohn, J.L. and Fischer, A.E.: POSTGRAD. MED. 5:20-26 (Jan.) 1949.

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